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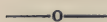
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HAIG, ALEXANDER, M.A. &
M.D. (OXON.), F.R.C.P.

BRUCE, ALEXANDER, M.D.

MOTT, F. W., M.D.

HEAD, HENRY, M.A., M.D.

TURNER, WILLIAM ALDREN,
M.D., F.R.C.P.

MACKINTOSH, ASHLEY W.,
M.D.

MUIR, ROBERT, M.A., M.D.,
F.R.C.P.E.

WILLIAMSON, R. T., M.D.
(LOND.), M.R.C.P.

COLLINS, JOSEPH, M.D.

ALLEN, F. J., M.D. (CANTAB.).

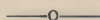
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BRAIN.

PART I., 1896.

Original Articles and Clinical Cases.

THE CHROME-SILVER METHOD. A STUDY OF THE CONDITIONS UNDER WHICH THE REACTION OCCURS AND A CRITICISM OF ITS RESULTS.

(Being the Presidential Address to the Neurological Society for 1896.)

BY ALEX. HILL, M.D.

Master of Downing College, Cambridge.

THE silver-method, discovered by Professor Golgi, of Pavia, has thrown more light upon the structure of the central nervous system than all other methods, microchemical, physical, and experimental, put together. In the address which Professor Schäfer delivered as President of this Society, in 1893, he gave a masterly account of the logical effects which these discoveries must have upon our views as to the constitution of grey matter and as to its mode of action as an apparatus for the distribution of nerve impulses.

The modifications in our hitherto accepted beliefs upon which Professor Schäfer insisted were chiefly of a negative character, but there was enough of reconstruction to show that no question is of such burning interest as this to anatomists, physiologists, and psychologists, or shall we say, in one word, to neurologists.

In 1865, Deiters pointed out the fundamental distinction between the axis-cylinder process and the protoplasmic pro-

cesses of a nerve-cell. In 1871, Gerlach gave expression to the usually accepted view as to the structure and mode of action of the nerve-machine. Gerlach's theory was extremely simple, and yet it made all the processes of nerve activity, from reflex action to thought itself, easily explicable. We could, in fancy, follow the afferent impulse as it traces its way along lines of least resistance through the network of nerve-strands to the appropriate efferent fibre: nay, we could see impulses which had sunk out of consciousness, taking a dynamic attitude within the cells in which they had lain dormant, and starting on a fresh voyage through the network, coming into relation with other impulses similarly liberated—combining, disparting, interweaving the tissue of a dream or a thought. The apparatus by which these processes are carried out was supposed to consist of afferent fibres ending in a network which receives also the protoplasmic processes of the nerve-cells, of which the axis-cylinders become nerves. Nothing could be simpler or more satisfactory. Without some such conception, the brain, as a mechanism, becomes unthinkable, for it bears no relation in character to the products of its own activity.

But this simple faith received a terrible shock when, in 1875, Golgi introduced his method for colouring the brain with silver. This method revealed the existence of branching systems hitherto unsuspected—the collateral ramifications of axis-cylinder processes and of posterior root fibres—and, at once, our views as to the way in which grey matter does its work fell into a state of chaos.

Golgi himself could not get free from the prejudices in which he had been brought up, but he deduced from his facts the only conclusions which were logical from his point of view. He had discovered a second *plexus* formed by the collaterals. He did not want two; and, moreover, he traced the protoplasmic processes of the cells into connection with blood-vessels and ependymal cells. Hence he was justified in throwing Gerlach's plexus overboard, and accepting, as a reflex apparatus, in its stead, the plexus of collaterals. Protoplasmic processes he relegated to an ancillary position, for he regarded them as nutritional; the roots

by which cells suck up the nourishment they require both for themselves and for the long fibres to which they give rise.

Fancy the grand branching system of a Purkinje-cell reduced to the level of a vegetable root! The position seemed intolerable, and besides we have no analogy for supposing that any animal cell needs such a system of roots, however extensive may be the processes of the cell body which look to it for their nourishment, or however important their functions. Indeed, it might be pointed out that the nerve-cells which are responsible for the nutrition of the longest fibres of all, the cells of the root-ganglia, are described by Golgi himself as destitute of this apparatus for collecting nutriment. Again looking at the question from another point of view, the processes of the granule cells of the cerebellum seem to lose their meaning if the protoplasmic processes of the Purkinje-cells are incapable of collecting impulses; so, too, in the olfactory bulb the glomerular protoplasmic processes of the mitral cells seemed clearly to be the chief centripetal conductors, and not to play a subsidiary part as Golgi suggested. Then the axis-cylinder processes of the large cells of the anterior horn of the spinal cord do not carry collaterals; in this case, therefore, impulses carried to the cord by posterior roots or by descending pyramidal fibres can only be transferred to the efferent root fibres *viâ* the protoplasmic processes of their cells of origin.¹ These and a variety of other reasons justified histologists in rejecting the nourishment-collecting theory of the function of protoplasmic processes.

The changes in our views as to the mode of working of grey matter are sufficiently startling, however, even though we do not classify cell processes as functional and vegetative. With regard to many of the features revealed by the chrome-silver method we have no physiological theories at all, and some seem to be incapable of explanation. From

¹ Kölliker ("Zur feineren Anatomie des centralen Nervensystems," II., *Zeitsch. f. w. Zool.*, li., p. 27, 1891) says that he cannot find collaterals on the axis-cylinder processes of motor cells. I also have searched for them in vain. Golgi figures them. Ramón y Cajal says that he observed them both in the rat and in the dove. We may therefore conclude that they are present exceptionally.

every point of view, therefore, it is essential that we should come to understand the conditions under which this method works, and learn how far we are to trust its results.

The suspicion with which we approach it would be unwarrantable in the case of almost any other method; but in many ways this method seems to court suspicion. To begin with, it is so coarse, from the point of view of modern histological technique, that we can say without any want of gratitude to the histologist who gave us this marvellous process, that it can have been discovered only by accident. A block of tissue soaked in bichromate of potassium, or in a mixture of bichromate of potassium and osmic acid, is dropped into a solution of silver-nitrate. A copious precipitate of bichromate of silver of course occurs. We should have expected that this precipitate would be uniformly distributed throughout the mass, whereas we find that the bichromate of potassium leaves the mass more quickly than the nitrate of silver enters it, and that after all the chrome-salt, which is in solution at the time, is precipitated, the nitrate of silver continues to penetrate and is reduced by certain cells and fibres in the interior of the block. The addition of a very little formic acid (about 1 to 300 of the bichromate and osmic acid mixture) delays the precipitation of chromate of silver, inducing it to crystallise out instead, and so favours the entrance of the silver-nitrate; as a result the interior of the block is freer from granular deposit than when nitrate of silver alone is used.

The cells and fibres which reduce the silver-salt are marked out with marvellous delicacy and present figures of great beauty, but the success of the method depends upon its failure—its failure as a stain. If the colour were to be taken by all the elements, or even by all the elements of one kind, the result would be an impenetrable black mass. It is because it picks out one cell in fifty, or one in a hundred that a picture is obtained which conveys information as to the structure of the tissue.

The results obtained by this method are so conspicuous and diagrammatic that no special skill in microscopy is needed for their recognition. So diagrammatic are the figures that

those who first used the method thought it quite unnecessary to copy them. More effective pictures could be made by merely imitating their general style. For a long time histologists were in doubt as to how far the artist followed nature, and how far he might be said to keep step with, or even to precede, her. These jet-black figures, on a tawny ground, are particularly suitable for photography; and this, as it seems to me, is by far the best way of reproducing them. They should only be drawn by hand when, as is not infrequently the case, the sections are too thick to allow of their photographic reproduction.

I have no intention of recapitulating the results obtained by Golgi and his pupils, by Ramón y Cajal, Lenhossék, Kölliker, Retzius, Van Gehuchten and others who have used the method with conspicuous success. Exhaustive, analytical, and bibliographical notices are accessible to everyone.¹ My endeavour has been to ascertain the CONDITIONS UNDER WHICH THE METHOD WORKS; THE NATURE OF THE REACTION; and, incidentally, THE VALUE OF ITS RESULTS. It has made startling revelations. How far may they be trusted?

What is the nature of the reaction? Why does it select certain cells or fibres and neglect the rest? Are there any cases in which the results obtained by Golgi's method are incompatible with those obtained in other ways, and if the two results cannot be reconciled, which is to go to the wall? These are the problems which I set myself to work out, and I may as well say at once that I am as far from knowing the answers as when I started. I can only hope that the publication of my work may save some other investigators trouble.

¹ Cf. v. Lanhossék, "Der feinere Bau des Nervensystems im Lichte neuester Forschungen," Berlin, 1893. Kölliker, *loc. cit.*; also "Handbuch der Gewebelehre des Menschen," vol. ii.; "Elemente des Nervensystems," 1893. Retzius, "Biologische Untersuchungen," *Neue Folge*, iii., iv., v., vi. Cajal, "Les nouvelles idées sur la Structure du Système Nerveux," trans. by Azoulay, 2nd edition, Paris, 1895. Van Gehuchten, "Le Système Nerveux de l'homme," Liège, 1893. Aldren Turner, "On recent applications of Golgi's Method to the Study of the Nervous System," *BRAIN*, 1893, vol. xvi., p. 259. Schäfer, Quain's "Anatomy," vol. iii., part 1, 1893. Obersteiner, "Anleitung beim Studium des Baues der Nervösen Centralorgane," 3rd edition, Leipzig, 1896.

Method.—The conditions necessary for success, as laid down by those who have chiefly used the method, may be summed up as follows :—

- (1) The subject should be a young animal or an embryo.
- (2) The tissue must be as fresh as possible.
- (3) It must be cut into thin blocks, not more than 2 mm. thick.

(4) During hardening, the bottles must be kept in the dark¹ and warm.

(5) If embedded in celloidin, the embedding process must not occupy more than an hour (for fear of destruction of the stain by absolute alcohol), and the celloidin should be set with 70 per cent. alcohol, and cut under alcohol.

Probably all these precautions are desirable under certain conditions, but that none of them are essential is shown by the photograph (fig. 1) from one of the most successful preparations I have ever made. It was taken from the brain of a man who had been dead for more than twelve hours; it was hardened in the cold of winter, kept in the light, embedded in celloidin in the ordinary way (the process extending over two or three days), and cut frozen on a Swift's microtone.

We are still so far from understanding the nature of the reaction, that all variations of procedure are possible sources of instruction. My object being not to get good specimens, but to determine the conditions under which the reaction occurs, I endeavoured to ascertain the limits within which the process may be varied without abolishing the reaction, and have noted the effects of the several modifications.

First, as to *material*.

(a) *Age of Subject.*—For general purposes young animals are desirable. The method succeeds well with embryos after about half-time (in the case of the calf, horse, and other large animals); new-born animals give excellent preparations of cortex cerebri, spinal cord, &c.; nevertheless, the most comprehensive results are obtained with animals of some days old, *e.g.*, kittens of three to twenty days, puppies

¹ In his later publications, Ramón y Cajal says that it does not matter whether the preparations are kept in the dark or in the light.

of a fortnight, rats of eight to twelve days, hedgehogs of nine days, &c.

For the cells of the cerebellum and cortex cerebri, I prefer a full-grown animal.

For the collaterals of posterior root fibres, it seems to be necessary to use a young animal: but this rule does not hold good for the axis-cylinder collaterals of pyramids and Purkinje cells. I have obtained beautiful preparations of axis-cylinder collaterals from the brains of full-grown hedgehogs.

Ramón y Cajal and Van Gehuchten recommend the use of embryos or very young animals on the ground that it is the non-medullated, or not yet medullated, fibres that give the reaction; but neither of the histologists just quoted states explicitly, so far as I am aware, that medullated fibres will not take the stain. This is a matter to which I have given some attention, in the hope that it might throw light upon the nature of the reaction, and I have come to the conclusion that although embryonic fibres and non-medullated fibres give it best, it is also possible to obtain the reaction through the medullary sheath. When Weigert and Golgi preparations are compared, it is found that the stainings do not tally. In the cortex cerebri or cerebelli, for instance, the Weigert stain follows the fibre close up to its cell: while in the Golgi preparations, the axis-cylinder process is stained black for a long distance. Again, in the white matter of the brain, *e.g.*, the internal capsule, it often happens that the silver colours great masses of fibres where we have no reasons for supposing that non-medullated fibres preponderate.

(b) *Freshness*.—It is especially desirable for this method of preparation that the tissue should be fresh, since the mixture of osmic acid and bichromate of potassium penetrates very slowly. If a block of two millimetres in thickness is taken out of the hardening agent after twenty-four hours and cut across, the centre will usually be found to be perfectly uncoloured, and it is easily to be understood that many *post-mortem* changes may have occurred in the interval, especially if the bottle has been kept warm.

I prefer to open the chest of the animal before the heart has ceased to beat, and then, having thoroughly washed out the blood-vessels with salt solution under suitable pressure through a canula inserted in the aorta, to inject a considerable quantity of bichromate of potassium into the circulation.

In my first attempts to inject warm bichromate of potassium into the living vessels, I found that the small arteries closed so vigorously against the irritant fluid that it was impossible to drive it through. By the addition of 1 per cent. of ordinary fermentation (ethylidine) lactic acid to the salt-solution I succeeded in producing such a paralytic dilatation of the small vessels, as judged by the free outflow from the cut jugular vein, that when the bichromate solution was used it flowed through with the greatest facility. Tooth has found that the injection of morphia after the salt-solution is equally effective in producing paralysis of the arterioles.¹ Sometimes I use the bichromate and osmic mixture for the injection, but it does not appear to possess any advantage over the bichromate alone; while in the quantity I use (250 cc. of the mixed osmic and bichromate) the cost is considerable and the smell extremely disagreeable.

This question of the *penetration of the hardening fluid* is, I believe, a factor of great importance in determining the subsequent reaction.

The excellent results which are to be obtained with the hedgehog's brain seem to be due, at any rate in part, to its unusual firmness and permeability; the difficulty in staining the human brain, to the closeness of its texture.

The success of the method when applied to young animals appears to me to depend partly upon the ease with which the hardening fluid, and, subsequently, the silver-nitrate penetrate.

Of much more consequence, as guiding us in forming an estimate as to the amount of reliance which we are to place upon the appearances presented by Golgi-preparations, is the fact that, in any part of the brain, the staining is more abundant and conspicuous when the tissue presents an open tex-

¹ Tooth, BRAIN, vol. xv., 1892, p. 400.

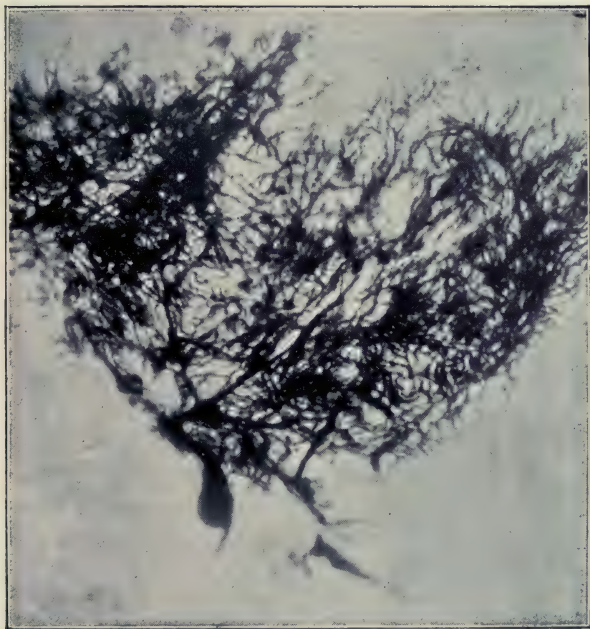


FIG. 1.

A cell of Purkinje from the cerebellum of a man aged 45, together with the branching system of a second cell.

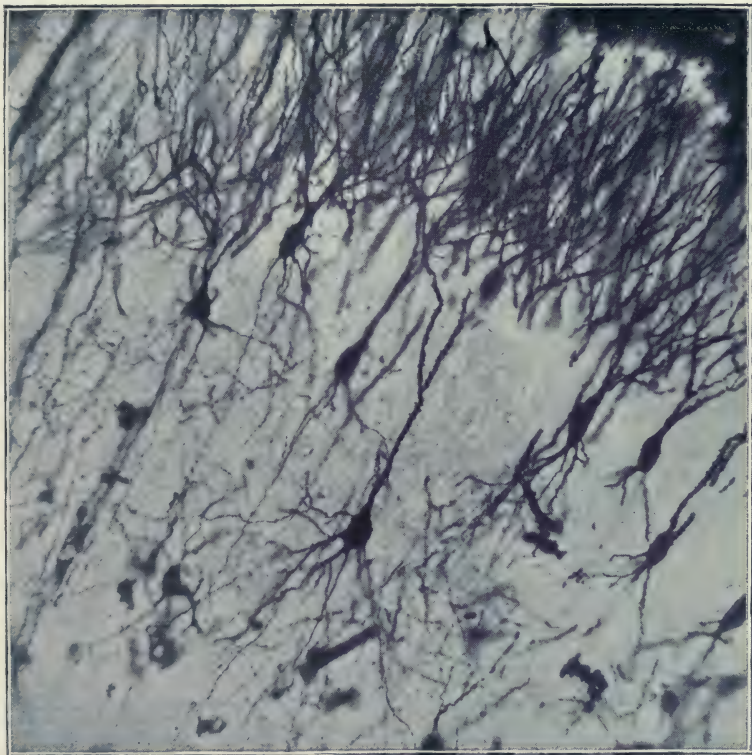


FIG. 2.

Adult hedgehog; cortex cerebri $\times 400$. The tissue was embedded in celloidin, thickened very gradually, the process extending over four days. A thin, flat section could be obtained in consequence. The thorns on the protoplasmic processes are well seen.



FIG. 3.

Skate (*Raia batis*); granular layer of the cerebellum, stained with the alum-carmin-Weigert method. The figure shows the nuclei of "granules" surrounding clumps of molecular substance. In the centre of the molecular clumps are seen the swollen axis-cylinders of the rosettes of Cajal's mossy fibres.

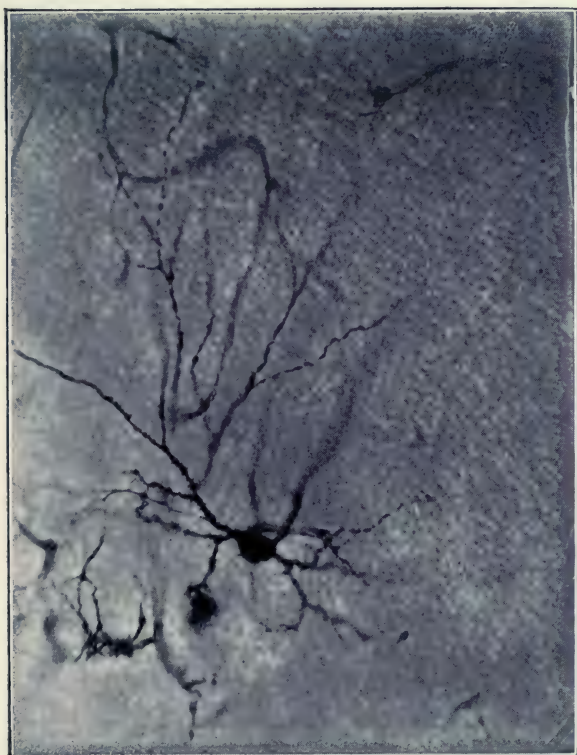


FIG. 4.

Adult hedgehog ; cerebellum $\times 408$. A single large cell of the molecular layer, with thorny protoplasmic processes and an axis-cylinder process from which a branch passes inwards to form a basket (only partially coloured) around a cell of Purkinje.

ture than it is where the tissue is dense. Many illustrations of this law may be cited; I will content myself with two which appear to me to be of the greatest importance: (a) In the cerebellum the thin sheet of tissue in which the bodies of the Purkinje-cells lie is extremely open, especially after the shrinking of the cells in the hardening agent. As a result we obtain in this layer an abundant staining of the fibrils which pass through it. Various appearances, often completely limited to this thin stratum, are seen in consequence. Sometimes the sheet is pierced by multitudes of vertical fibrils, the centrifugal fibrils of the "granules." At other times, either the branching processes of the tangential cells of the molecular layer or the branching processes of penetrating nerve-fibres are abundantly stained as they lie in this sheet.

The fascia dentata affords another illustration of the limitation of the stain to a layer. It usually happens, in a transverse section of the cerebrum, that we find that the cells of the granular layer of the fascia dentata, where it lies near the surface, are beautifully stained; the underlying cells of the nucleus fasciæ dentatæ may or may not be stained; the small pyramids of the granular layer where it lies at a distance from the surface are hardly ever stained, but we see in their place a quantity of penetrating fibres (fig. 7). The difference in staining of the fascia dentata in its two parts can only depend upon the relative freedom of access afforded to the reagents. Usually the fibres in the deep-lying fascia dentata are absolutely limited to this layer which is marked out by the total absence of stain on either side of it. It seems to me difficult to account for this limitation of the reaction on any hypothesis other than the one I have put forward, viz., that the reaction occurs most readily where the tissue is loosest in texture.

(2) The occurrence of the reaction does not appear to depend upon the tissue being fresh, for it can certainly be obtained a considerable time after death—at least two days in cool weather. If it were not for the slowness with which the hardening fluid penetrates, good results might, I believe, be relied upon with any tissue in which putrefaction had not already set up.

On the other hand, my experiments in injecting tissues with bichromate during life seem to show that absolute freshness has a good deal to do with the character of the results obtained. When the injection has been successful—and it is easy to tell from the appearance of the unstained nuclei whether this is the case—not only are deeply lying structures, such as the corpus striatum, stained before the pyramids, but all the cells show the most remarkable clearness of outline. The processes of the cells are sharp and clear cut, and there is in such preparations as I have made a total absence of “thorns” or “mossiness.” To this I shall refer later on.

(3) *Size of the Block*.—As observed by everyone, the amount of osmic and bichromate mixture used to harden the brain should be large relatively to the amount of tissue, but the brains of different animals vary immensely in their capacity for absorbing the reagent. Of all the animals I have used none allows the reagent to penetrate so easily as the hedgehog. A complete transverse section of the brain of the young hedgehog is easily stained throughout its whole extent, giving a perfect result, even though the slab of tissue be 3 or 4 mm. thick. I attribute this penetrability to the peculiar texture of the brain which, when fresh, is white, and so firm as to be convenient to handle.

If the blood-vessels are injected with warm bichromate of potassium, the amount of tissue hardened in one block may be almost indefinitely increased, since the nitrate of silver finds its way through any thickness of tissue, giving apparently a better result the farther it has to go.

(4) *Duration of Hardening*.—In one paper (*Rivista trimestriale de histologia*, March, 1889, p. 104) Ramón y Cajal gives the following general rule:—

For neurogleia, harden 12-20 hours.

„ nerve-cells „ 20-24 „

„ nerve-fibres „ 24-36 „

In another place (*La Cellule*, vii., 1891, p. 130) he amplifies his directions by pointing out that the time needed depends not only upon the structure which it is

desired to show, but also upon the kind of animal and its age. For example: "In the embryos of mice, rats and rabbits the cells impregnate in a very uncertain fashion; on the other hand, the epithelial elements, vessels and fibres colour constantly, provided the duration of the hardening is not prolonged beyond two to three days. The duration of the hardening of the blocks in the osmio-bichromate mixture ought to be prolonged to two, three to five days when dealing with new-born mammals of a certain size (rabbit, guinea-pig, cat), et, à plus forte raison, s'ils sont âgés de 8 à 15 jours."

"To succeed in colouring the cells of the molecular layer in the rabbit (eight days old) the hardening must be prolonged for nearly five days; while the collaterals of the white substance demand six or seven."

Lenhossék ("Der feinere Bau des Nervensystems," Berlin, 1893, p. 6) gives as the most favourable time for the human spinal cord—

Neurogleia, 2-3 days.

Nerve-cells, 3-5 days.

Nerve-fibres (collaterals), 5-7 days.

When I commenced this series of tests it was my intention to compare Golgi-preparations of all parts of the brain with sections stained according to my modification of Weigert's hæmatoxylin method.¹ I wished to ascertain whether the silver colours tissue only, or whether, as was suggested by Rossbach and Sehrwald, in 1888, it colours instead of, or in addition to, the neurogleia and nerve-tissue, lymph, or lymph-deposit, or lymph-coagulum which surrounds the nervous structures. I was soon convinced that

¹ My modification of Weigert's method depends upon a previous staining of the tissue *en bloc* in carmine-alum. The carmine-alum is made by boiling an excess of both carmine and alum in a porcelain dish for three or four hours, water being added from time to time. The fluid is allowed to stand for some hours, and then filtered. Blocks of tissue are soaked in carmine for twenty-four hours; sections are then made and stained according to Weigert's hæmatoxylin method; decolouration may be effected with either ferridcyanide of potassium or permanganate of potash, the solution being in either case of about half the strength usually recommended. All nerve-cells and their processes are stained black; medullated fibres are stained blue, and non-medullated fibres black (*cf.* the photograph, fig. 13).

while the black reduction-substance may lie entirely outside the nerve-elements, forming a case for them, or may both surround and penetrate the nerve-elements, it is, in the most successful preparations, limited to the nerve-elements themselves. I therefore abandoned the comparison with Weigert preparations. For the sake of the comparison, however, all my earlier preparations were made under rigidly uniform conditions, the tissue remaining in the hardening fluid for exactly five days. Among these preparations I find admirable examples of cells of all kinds, of granules, fibres, collaterals, &c.; and hence this test, as well as my recent experiments in which I have varied the time of hardening within the widest limits (one day to two months), fail to establish anything like a definite relation between length of hardening and elements stained. Preparations of the cortex of a twelve-day rat, which had remained in the bichromate and osmic mixture for two months, exhibit pyramidal cells with their axis-cylinders and collaterals with exceeding beauty and completeness.

Ramón y Cajal says that some elements may be lost because the hardening in bichromate of potassium has been too prolonged. Of this I have never seen proof.

That pieces of tissue which after three, four, or five days' hardening give very little reaction may give a good result at the end of ten days or a fortnight I am fully convinced, but I cannot lay down any law as to the form which the improvement will take.

The variability of the result leads us to hope that when we understand the conditions under which the reaction occurs we shall have in our hands a method with which to produce at pleasure a wide range of effects, and it is very greatly to be hoped that someone will undertake an exact series of observations upon its time- and temperature-relations.

Prolonged immersion in silver-nitrate does not appear to affect the reaction.

(5) *Temperature*.—I have hardened the tissues at every temperature from the cold of winter to 45° C. Warmth hastens the reaction, but it does not, so far as I can determine, affect its quality.

(6) *Light*.—As observed elsewhere the reaction occurs in bright sunlight as well as in photographic darkness.

(7) *Celloidin*.—The rules for embedding, as laid down by various observers,¹ present considerable discrepancy although all agree that the sojourn in the alcoholic solution of celloidin should be as short as possible. I have prolonged it up to four days without any perceptible deterioration, and with the obvious advantage that the celloidin when set and cut does not pucker but lies perfectly flat on the slide. The accompanying photograph (fig. 2) is from a section of a block placed for some hours in a mixture of alcohol and ether, to which celloidin was added in gradually increasing quantities until at the end of four days it was ready to set. It is a matter of great importance, if celloidin is to be cut frozen, that its constitution should be uniform. I am in the habit of gradually adding dry celloidin to the alcohol and ether in which the block is soaked. Not only does this ensure penetration, but the sections when cut lie perfectly flat. It is just as easy to make a section of the whole brain, either transverse or longitudinal, as to cut a minute piece.

Variations in the Hardening Agent.—There is little latitude for variation in this direction. A chromate of some kind must be used, and the potassic bichromate seems to answer better than other salts. Ammonic bichromate gives the reaction, but whilst the cells come out very clearly, and the fibres are clearer than with potassic bichromate, the finer structures are not seen.

Salts of chromium, other than the chromates, do not give the reaction. Chrome-alum, for example, which for some purposes is an excellent hardening agent, is, for this method, perfectly useless.

Osmic acid expedites the hardening, but does not take part in the reaction; indeed, it may be replaced by formaline

¹Ramón y Cajal: "The pieces can be quickly embedded in celloidin or paraffin." Kölliker: "One hour in absolute alcohol and one hour in celloidin." V. Gehuchten: "Fifteen to twenty minutes in 96 per cent. alcohol, a quarter of an hour in absolute alcohol, a quarter of an hour in thin celloidin. The block is fixed on cork with celloidin, and immersed for a short time in 70 per cent. alcohol." Lenhossék recommends five minutes in a moderately thick celloidin solution; placing the object in elder-pith; setting celloidin with 80 per cent. alcohol, and cutting under alcohol.

(4-6 per cent.), as Durig¹ has shown. There is, therefore, no need for such a cumbersome name as bichrom-osmio-silver method (Retzius): the "chrome-silver method" best expresses the reaction.

Variations in the Silver-salt.—The only experiments I have tried have been in the direction of a salt more easily reducible than the nitrate.

(a) Silver-acetate gives the reaction, but does not pick out the finest markings, and is usually accompanied by a great quantity of deposit.

(b) Silver-nitrite, saturated solution, *i.e.*, about $\frac{3}{4}$ per cent. (with the addition of formic acid about 1 to 1,000 parts of the nitrite solution), gives an excellent result. The reaction seems to differ from the reaction obtained with the nitrate in certain important respects, and I am hoping that this will prove a useful modification of the process. So far as my observations go at present, they lead me to think that the nitrite gives a better reaction with the extremely minute fibrils than the nitrate; it gives a good preparation of collaterals; it shows granules lying in an abundant feltwork of fibrils; the branches of protoplasmic processes are more numerous, and their "thorns" more distinct than when nitrate is used.

The Nature of the Reaction.—The term "stain" has been generally abandoned since it is clear that the reaction which occurs when a tissue soaked in bichromate of potassium is plunged into a solution of nitrate of silver is not comparable to a process of staining by an organic stain. Ramón y Cajal speaks of the black colouration as a "deposit of chromate of silver."² Van Gehuchten refers to it as a "reduction." In fortunate sections the chief colouration is black, without a tint of either green or red. There is almost always in the same sections a red-brown colouration of blood-vessels and other structures similar to the effect produced upon fresh tissue (especially intercellular "cementing substance") by the action of silver-nitrate alone. The extreme blackness

¹ Durig "Das Formalin als Fixirungs mittel anstatt der Osmiumsäure bei der Methode Ramón y Cajal's," *Anat. Anz.*, x., No. 20.

² Ramón y Cajal, *La Cellule*, vii., p. 131 and elsewhere.

of the black deposit makes it quite clear that it is not a chromate of silver. It must be a reduced salt (sub-salt) of silver. That it is not a metallic oxide or a salt reduced so far as in photography is shown by the solubility of the deposit in hyposulphite of soda or dilute ammonia.

One of the most singular peculiarities of the deposit is its rapid disappearance when the section is covered with a coverslip. All the "staining" may disappear, if the section is mounted in ordinary Canada balsam, in four or five hours, although masses of black deposit remain permanently. Since most of the gum-resins have a weak oxidising action, the disappearance of the colour might be attributed to the oxidation of the reduced silver-salt. If, however, the process is watched under the microscope, appearances are noticed which indicate that the change is due, as Sanassa suggested, to physical causes—at any rate, in part. The colour first leaves the centre of cells or fibres. Dark brown granules then appear in the matrix in the vicinity of the structure which is losing its stain, and it would seem as if the deposit is withdrawn from the tissue by a mechanical force; perhaps its displacement is due to currents set up by the drying of the balsam at the edge of the coverslip. I find that when a slowly-setting turpentine balsam is used without a coverslip, the colour stays in the section, even though the balsam about it may remain liquid for days. On the other hand a large part of the stain is permanently retained in a covered section if the balsam has been evaporated until it sets in a brittle mass, and then re-dissolved in xylol or chloroform. Preparations made with nitrite are also comparatively durable, and the fact that hyposulphite of soda does not destroy all the colouration shows that the reduction is more complete in some parts (particularly the cells and their protoplasmic processes) than in others, and holds out a prospect that a modification will soon be discovered which will ensure the permanence of the preparations when covered with a cover glass. Various methods have been suggested for fixing the silver-deposit—methods which naturally occur to every photographer. It may be toned in an alcoholic solution of chloride of gold

(Obregia¹), or converted into a bromide which is subsequently reduced by light (Greppin²), or the reduction may be completed by sodic sulphide, eikonogen, or any other developer, and it may be mounted beneath a coverslip with or without subsequent toning with gold or with platinum. It is very easy in most cases to obtain a preparation which is permanent, although covered, but there is always, so far as my experience goes, a loss of delicacy. If reduction is completed by a developer, the ground-substance, which still retains traces of silver-nitrate, either free or in chemical union with proteids, is darkened. If development is not completed before toning, some of the finer markings are lost.

I find that good covered preparations may be obtained by staining in fuchsin, with subsequent treatment with picric acid in spirit, and washing. Such preparations are usually too dark, but may be of use in showing the position of the black structures in relation to other cells and fibres.

What is the nature of the chemical substance which, when united with a chrome-salt, reduces the silver? It might be *doubted whether a chrome-salt is necessary*, but such experiments as I have tried seem to show that it is. All my attempts to reverse the process, using the silver first and the bichromate of potassium afterwards, have failed, even when nitrate of silver or lactate of mercury was passed through the cerebral blood vessels. Again, if acetate of copper is used after the chromate it prevents the reaction unless the block is returned to the chromate for a time, in which case it takes the stain as usual.

The fact that the preparation for the silver-stain is effected by a chrome-salt suggests that the reduction or non-reduction of the silver-salt depends upon the extent to which the chromate is fixed by light. Chrome-salts, as is well known, are profoundly changed by the action of light. If a gelatine plate be immersed in a chromate and then exposed

¹ Obregia, "Fixirungsmethode der Golgischen Präparate," *Virchow's Arch.*, cxxii., 1890, p. 387.

² Greppin, "Weiterer Beitrag zur Kenntniss der Golgischen Untersuchungsmethode der Centralen Nervensystems," *Arch. f. Anat. und Physiol.*, Suppl. Heft., 1889, p. 55.

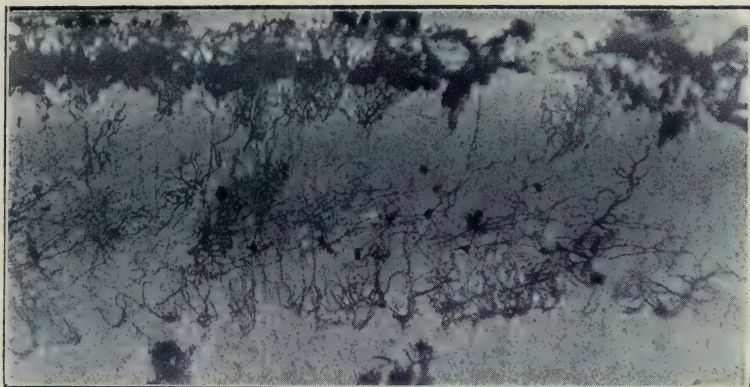


FIG. 5.

Adult cat; cerebellum. Large cells of the molecular layer are seen forming baskets about the cells of Purkinje. The cells of Purkinje are not coloured, but portions of their branching systems are shown at the top of the molecular layer.

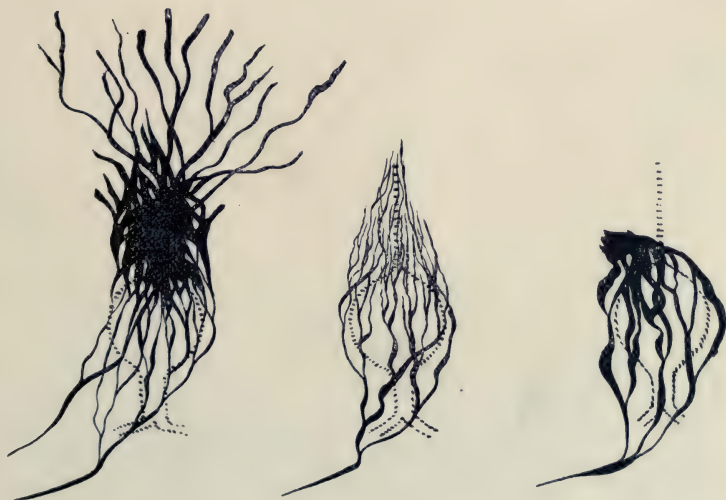


FIG. 6.

Diagrams of the three types of "basket" found in the cerebellum:—

- (a) The appearance most commonly observed.
- (b) The tassel of separate fibres figured by Cajal.
- (c) The tassel ending in a black mass beneath the cell of Purkinje and fibres streaming off from this mass into the granular layer.



FIG. 7.

Adult hedgehog ; fascia dentata hippocampi. Showing on the superficial side of the fascia dentata the colouration of cells, while only fibres are stained in the part farthest removed from the surface. This indicates the effect which the texture of tissue has upon the chrome-silver reaction.

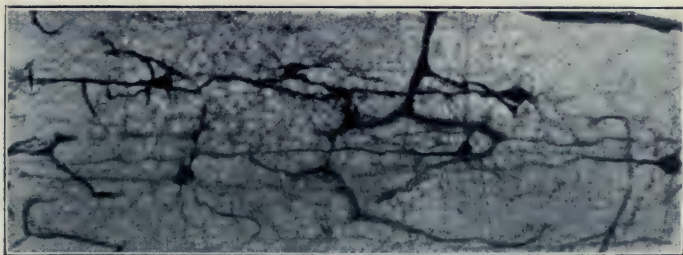


FIG. 8.

Rat, 4 days old ; cortex cerebri. The apical process of one pyramid runs into the base of another more superficially placed.

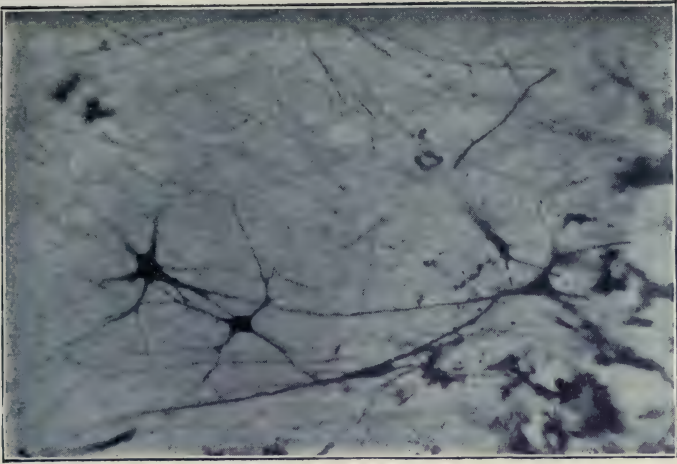


FIG. 9.

Calf, six months (*Octus*), spinal cord. Showing the connection of anterior horn cells by stout processes.

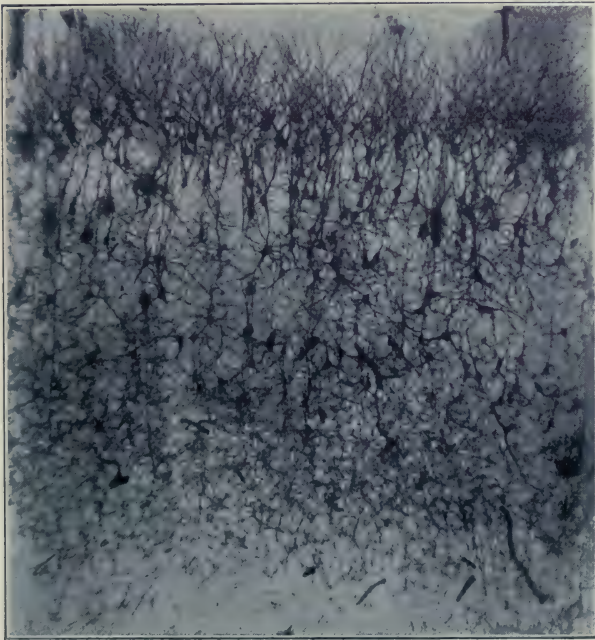


FIG. 10.

Adult hedgehog; cortex cerebri. The vessels were injected with warm bichromate and osmic mixture. The protoplasmic processes of the pyramids are destitute of thorns.



FIG. 11.

Adult hedgehog; cortex cerebri. Showing the junction of Meynert's second and third layers. The protoplasmic processes of the pyramids are thorny.

to the light (as in one of the methods used in preparing plates for photo-printing), the chrome-gelatine compound upon which the light falls is rendered insoluble. It seemed possible that light penetrates the blocks of brain immersed in bichromate of potassium and reaches some cells while it is blocked out from others, but various experiments which I have made show that this is not the explanation. For instance, I divided a brain into two halves; put one half, in suitable pieces, into a bottle of bichromate and osmic which was enveloped in black paper and kept in the dark throughout all stages of the preparation, only bringing the pieces into the light when they were placed on the microtome. The other half of the brain was exposed throughout all stages of its preparation to the full light of a west window. The reaction succeeded equally well in the two cases, although the results were somewhat different. It seems unnecessary to record the differences in detail, since they may be attributed either to the caprice of the method, or to the fact that the bottle exposed to the light was warmed by the afternoon sun while the other bottle was kept at a uniform temperature.

The fact that in one part of a section the stain will mark out the nerve-cells and in another the neurogleia, precludes us from looking to any chemical body peculiar to nervous tissues as the attractive substance. The singular way in which in all sections one element, whether large nerve-cell, small nerve-cell, or nerve-fibre is picked out, while a dozen, a score, or perhaps hundreds of precisely similar elements are passed by, suggests that the condition of nutrition of the cell determines its reacting power.

Does the reaction depend upon the condition of nutrition of the elements?—To test this by exhausting one part with repeated stimulation while another part remained at rest was a natural suggestion; but the fact that the method so often selects the neurogleia cells led me to doubt whether any result would be obtained. It seemed better to try to affect the nutrition of the whole of the brain at once and profoundly. With this end in view, I determined to wash the blood out of the brain, and to compare this starved

brain with one removed from an animal before the cessation of the circulation.

(1) A stream of salt solution (.6 per cent.) at the temperature of the body, was passed into the aorta of a puppy for more than half-an-hour. The water left the jugular vein quite free from colour; but the brain took the silver reaction in the usual way.

(2) In the case of a second puppy, 1 per cent. of ordinary fermentation (ethilidene) lactic acid was added to the salt-solution. It was passed for more than half-an-hour, and appeared from the swelling of the body and the rapidity with which it left the pressure bottle to cause great dilatation of the blood-vessels. The brain took the reaction as usual.

These experiments, 1 and 2, were repeated in the hedgehog, and other animals.

(3) After using (in the case of an adult hedgehog) normal salt-solution for a short time, the pressure bottle was filled with a 10 per cent. solution of NaCl. It flowed freely through the vessels for more than half-an-hour. The body of the animal became greatly distended. All the connective tissues were found to be filled with transparent colourless jelly, resembling vitreous humour. The brain was semi-transparent, soft, and very difficult to handle, remarkably different from the ordinary hedgehog-brain, which is particularly firm and white. Nevertheless it took the reaction as usual so far as the selection and rejection of elements is concerned, although the appearance of the elements was changed in certain details.

The results of this last experiment seem to me very remarkable. The strong salt-solution must have dissolved and carried away a great quantity of albumins and globulins. All "cement-substance" or other lymph-deposit must have been dissolved. The softness of the brain showed that it had lost much of its substance, and yet it reacted to the bichromate and silver in the usual way. It seems quite to exclude any state of nutrition as a factor in determining reduction; at the same time it makes it very difficult for us, unless by a process of elimination, to put our finger upon the chemical substance which, when combined with a chrome-salt, reduces the nitrate of silver.

In face of this result, I did not think it worth while to carry out the experiments upon the effects of CO₂, morphia, chloroform, &c., which I had in contemplation.

INTERPRETATION OF RESULTS.

In summing up the contributions to our knowledge made by Golgi, Ramón y Cajal says¹ that the researches of this *savant* have placed *hors de discussion* the following facts:—

(1) The free termination of the protoplasmic expansions of nerve-cells, as well as of the processes of neurogleial, or spider cells.

(2) The existence in the axis-cylinder of ramified collaterals.

(3) The existence of two kinds of cellular elements, distinguished by the behaviour of their axis-cylinders; the cells in which this process loses its individuality by ramifying without reaching the white matter, and the cells of which the nervous process, despite its numerous collaterals, preserves its individuality, and continues into a tube of the white substance.

I should add to this catalogue of the great changes which the silver method has introduced into current nerve-histology:

(4) The existence in various parts of the central and peripheral nervous systems of a form of nerve-ending, first described by Ramón y Cajal the pericellular basket work; with the physiological inference that nerve impulses act upon (or from) nerve *cell-bodies*, through the medium of a brush of fibres.

(5) The conclusion that axis-cylinder processes, and their collaterals, as well as protoplasmic process have free terminations; that there is no such thing as anatomical continuity of nerve-elements, with the physiological consequence that transference of impulses occurs *in distans*.

(6) The great prominence given in making schemes of the several cortical fields other than the cerebellar cortex, to large nerve-cells, their axis-cylinder processes, and

¹ *La Cellule*, vii., 1891, p. 126.

collaterals; these elements, together with certain free-ending fibres, being held to constitute the reflex mechanism, to the exclusion of the "granules."

These changes, introduced by those who have worked with the chrome-silver method into our conception of the central nervous system, are of such importance that the grounds upon which they are based need the fullest investigation. It is our duty to assume a sceptical attitude of mind towards the new histology, in justice to the investigators who patiently laboured in this field before the introduction of the silver method. We fully realise that Golgi's predecessors did their work with tools which now seem primitive, but they worked without tiring, absorbed in their zeal for truth, and they deserve that the results of their labours should be upheld until swept from the field by an overwhelming weight of argument.

The absence of uncertainty, the simplicity, one might almost say the ingenuousness, of the silver-preparations carry all before them. It is difficult to resist the belief that they tell the truth, the whole truth, and nothing but the truth. For the purposes of this address, however, I have accepted the position of counsel for the defendants, and I beg to be allowed to lay before this court certain considerations which tend to invalidate the testimony of the plaintiff.

I will sum up the evidences of untruthfulness which tend to shake our faith in the chrome-silver method under three heads:

(1) The deposit is liable to spread beyond the limits of the elements which chiefly attract it. In some cases it stains, not the cell or the fibre, but the wall of the lymphatic space which surrounds it.

(2) The amount and character of the reaction depends upon the texture of the tissue in which it occurs. It is, therefore, liable to stop short at the edge of a favourable zone, giving an incomplete picture of the elements which it colours.

(3) *a.* It never takes all the elements of any one kind. *b.* It seldom shows heterologous elements in the same section with sufficient completeness to enable us to trace their connections. *c.* It does not stain the whole of the substance

which belongs to a cell or a fibre, but only part, and this apparently the neuroplasmic not the conducting part.

We may now consider how far these three drawbacks to the method affect the credibility of the great conclusions which have been drawn from its results.

(1) *The Spread of Deposit*.—Of this we need say but little. Everyone who has worked with the chrome-silver method is on his guard against this danger. It is especially in the case of neurogleia that the deposit is apt to occur outside the cell. I am inclined to believe, to take an example from the neurogleia, that the extraordinary irregularities on the course of the central processes of Bergmann's cells of the cerebellum, as figured by Retzius,¹ are collections of deposit. It may also occur on nerve-elements; thus it serves to exaggerate the mossiness of Cajal's mossy fibres, although the existence of these "fibras musgosas" is now placed beyond dispute. In good preparations the branching fibres of this system are of exquisite delicacy and clearness of outline. In fig. 3 the thickenings at the points of division, which constitute the "rosettes," are shown by another staining method. In passing, it may be noted that it would be a great advantage if the term *rosette fibres* could be substituted for mossy fibres, since the thickened bunches which these nerves carry are unlike any kind of moss except, perhaps, certain lichens, and, besides, the term "mossy" is constantly and aptly applied to an entirely different appearance which distinguishes the protoplasmic processes of many large cells both nervous and epithelial.

The staining of the lymph-case in which cells and their processes lie is a phenomenon which has not received the attention it deserves. It is of very frequent occurrence in the cerebellum. With or without simultaneous staining of the cells of Purkinje, the wall of the space in which these cells and their chief processes lie is coloured, usually a full brown, but sometimes a very dark brown or even black. It is difficult to show this in a photograph, or even in a drawing, since the appreciation of the fact that one is

¹ Retzius, "Biologische Untersuchungen," *Neue Folge*, vi., 1894.

looking at a hollow case depends entirely upon variations of focus. I am, therefore, obliged to content myself with a verbal description. Owing, no doubt, to the fact that many fibrils pass through the case, its wall is always sculptured in an elaborate but variable pattern. It frequently extends, for a short distance, outwards along the course of the fibrils which pierce it. Of course it follows the outlines of the cell and its chief protoplasmic branches.

After all that has been said about Cajal's *fibres grim-pantes* (kletterplexus, climber plexus), and especially since they have been carefully investigated by Retzius, and their authenticity is vouched for by so competent an histologist, I hesitate to suggest that they are an illusion due to the silver-staining of the lining of the lymph-case which surrounds the Purkinje-cell and its processes. If I saw the preparations from which Cajal and Retzius derive their descriptions, it is probable that I should recognise at once that such an explanation is out of the question. I content myself, therefore, with asserting that the only preparations of my own which show the kletterplexus may be explained in this way.

The apparent continuity of the plexus with a nerve is a great difficulty in the way of this explanation; but, on the other hand, Cajal's description of the nerves as "*fibres épaisses à myéline, peu ou pas ramifiées à leur passage à travers les grains,*" would apply equally well to the neural processes of the Purkinje-cells.

(3) Of much more consequence than this basket-work surrounding protoplasmic processes are the baskets supposed to be formed by axis-cylinder processes and their collaterals about the bodies of large nerve cells. They have been described in the cerebellum, the retina, sympathetic ganglia, and elsewhere. The ending of a nerve in a brush of fibrils which form a basket to invest the body of a nerve cell is a new conception in histology which we owe to the Golgi method. Have we any alternative but to accept the picture which the silver draws as a correct and complete account of the structure? I hope to show that we are *not compelled to believe that the baskets which surround Purkinje-cells are the end-brushes of nerves.*

The cells which form these baskets are found in the deeper parts of the molecular layer of the cerebellum. They were first described by Golgi as cells of his second type. No cells are easier to stain (figs. 4 and 5). In many preparations their processes form so dense a felt-work that the molecular layer is impenetrably black, although no other structures are coloured. Their full extension can only be seen in antero-posterior sections, irregularly stained.

The oval cell-body, as seen in section, gives origin to five or six processes, some of which run towards the surface, while others turn towards the granular layer. One of the processes extends horizontally for a great distance (1 to 2 mm. in the cat) giving off at intervals branches which descend towards the Purkinje cells. This long process is rightly called the axis-cylinder process of the cell; but it will be noticed (*a*) that although it is thin, round, and well-defined at its origin, it has a curious tendency to thicken as it goes on; (*b*) that it never acquires a medullary sheath (or if it has such a sheath this does not interfere with its colouration); and (*c*) it is possible that some of the other processes also take part in the formation of baskets.

As its collaterals approach the cells of Purkinje, each of them divides into a brush (the "basket"), the filaments of which end freely, and eventually the axis-cylinder process itself terminates in the same way. Ramón y Cajal, v. Gehuchten and others are perfectly explicit as to the free ending; but it is with regard to this free ending that I venture to mistrust the silver method.

If a number of specimens of baskets are examined, it will be found that much the commonest appearance is the ending of the branches which invest the cell of Purkinje in an irregular patch or blob beneath the cell (fig. 6a). This appearance is set aside as incomplete. On the other hand, instances will be found, every here and there, in which, beneath the cell of Purkinje, the basket-work, after forming a dense tuft, streams off amongst the granules as separate fibres (fig. 6c). We have, therefore, three forms of basket to select from—(1) the loose basket-work ending in a

blob beneath the Purkinje-cell ; (2) the tassel of free-ending fibrils described by Ramón y Cajal (fig. 6b) ; and (3) the bunching together of the fibrils of the tassel and their continuation into the granular layer.

In deciding between these divers forms of basket, we need to give great weight to the influence which the open nature of the tissue forming the sheet of Purkinje-cells has upon the reduction of the silver-salt. It constantly happens that colouration is limited to this sheet—the fibres which traverse it being stained, while the molecular layer, on the one side, and the granular layer, on the other, are uncoloured. The weight which must be given to the mere physical texture of the sheet is well illustrated by the accompanying photograph (fig. 7), taken from a different part of the brain, in which the conditions are somewhat similar. Like the cerebellum on a small scale, the fascia dentata of the hippocampus consists of a layer of granules bearing a sheet of cells, each of which has an axis-cylinder process passing back through the granules and an elaborate system of branching processes which extend towards the free surface. The resemblance to the granular layer, the sheet of cells of Purkinje, and the molecular layer of the cerebellum, is very singular. This triple sheet of tissue is folded over the cortex margin or nucleus fasciæ dentatæ, and I have found in numbers of preparations of this region which I have coloured with the chrome-silver method, that whereas in the part of the fascia dentata which lies on the surface, the cells and their processes are beautifully defined, and only their axis-cylinder processes are to be seen in the granular layer, on the other side of the nucleus fasciæ dentatæ, where the tissue lies at a distance from the surface, no cells are stained, but numbers of fine fibrils are seen streaming through the granular layer. Now, it cannot be doubted that these fibrils extend beyond the limits of the granule layer, although it is only in this layer that they stain—owing, as I think, to the looseness of the tissue.

In the cerebellum, it is clear that the cells of Purkinje leave but little space for the passage through the sheet in which they lie, of the vast number of fibrils by which the

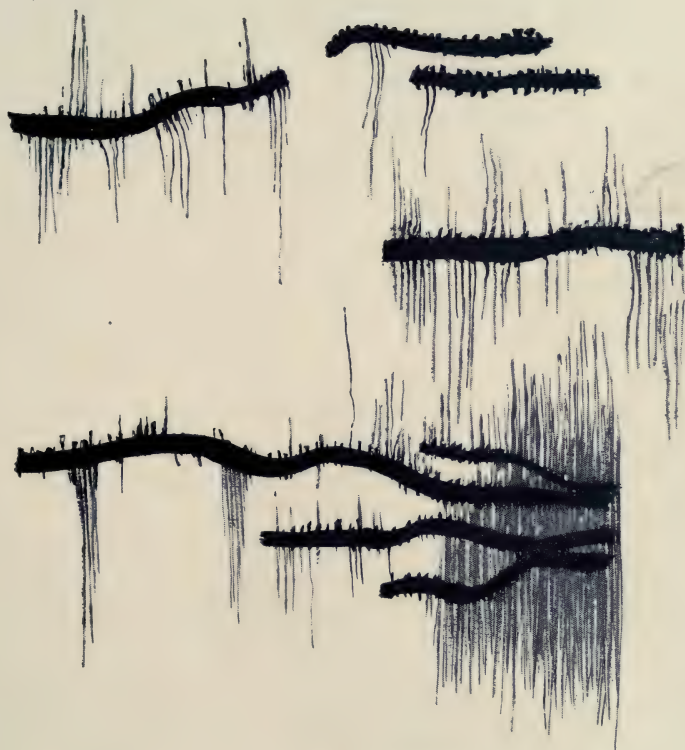


FIG. 12.

Skate (*Raia batis*); cerebellum. Sketch of the molecular layer, showing the union of horizontal fibres with the undulating protoplasmic processes of Purkinje-cells. In other parts of the preparation the processes were thorny. Here, horizontal fibrils of varying length take the place of thorns.

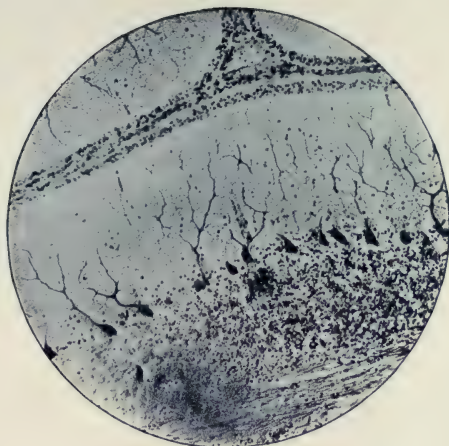


FIG. 13.

Lamb; cerebellum $\times 77$. The section was stained with the alum-carmin-Weigert method by myself, but I am indebted to Dr. Dallinger for permission to reproduce the photograph which appears on the frontispiece of his edition of Carpenter's "Microscope."



FIG. 14.

Rat, 4 days old; cerebellum. A group of granules with centripetal axis-cylinders. Two Golgi-cells are seen above the granules. The position of the cells of Purkinje is indicated by dotted lines.

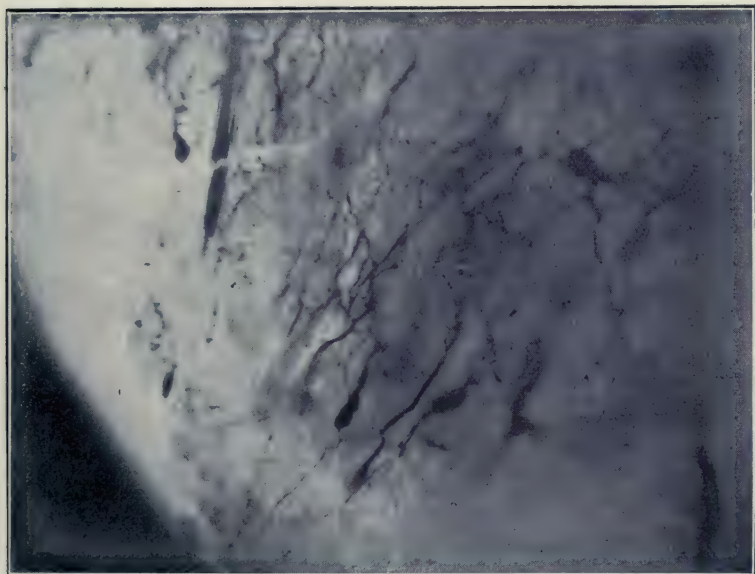


FIG. 15.

Rat, 4 days old; cerebellum $\times 398$. Photograph of a group of granules, with centripetal axis-cylinders and a Golgi-cell.

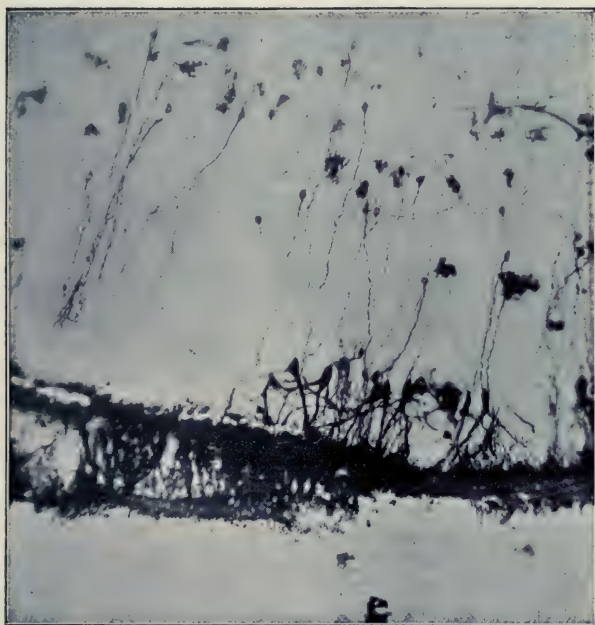


FIG. 16.

Kitten, 3 days old; olfactory bulb $\times 248$. Showing mitral cells and granules.

molecular and granular layers are united. These fibrils seem to me to arrange themselves in two sets: (1) the tassels of the "molecular" cells which place themselves around the cells of Purkinje, and (2) the axis-cylinder processes of the "granules" which occupy the intervening spaces. Owing to the peculiar texture of the sheet, and especially to the facilities for the passage of the silver nitrate which its radial striation affords, it comes about that the staining is much more complete here than elsewhere; BUT IT DOES NOT FOLLOW THAT BECAUSE THE FIBRILS OF THE MOLECULAR CELL TASSELS CANNOT BE FOLLOWED INTO THE GRANULAR LAYER THEY END HERE.

The object of my work is to determine the conditions and limitations of the chrome-silver method. I have no intention of giving in this paper a detailed description of any particular nervous organ; nor do I contemplate making a list of all the supposed revelations of the method which seem to me to require confirmation. These three illustrations from the cerebellum are selected merely on the ground that they are representative of the three main directions in which the *method is likely to mislead*.

(b) *Isolation of Nerve-Elements*.—The great conclusion deduced from observations made with the chrome-silver method is the discontinuity of the end-brushes of fibres and the ramified processes of nerve-cells. This anatomical conclusion is of profound interest to all who endeavour to explain the action of the nervous mechanism, whether physiologists or psychologists, since like the theory of which the anatomical basis has just been discussed—the theory that nerve-fibres end freely in contiguity with the bodies of nerve-cells—it is completely subversive of all existing notions of nerve-conduction.

Gerlach¹ described the posterior roots of spinal nerves as ending by ramification in the grey matter; the anterior roots as starting in cells; between the two a plexus formed by the union of the terminal twigs of the afferent fibres, the protoplasmic processes of the motor cells, and thirdly, the

¹ Stricker's "Manual of Human and Comparative Histology," translated by Henry Power, London, 1872, vol. ii., pp. 327 *et seq.*

protoplasmic processes of smaller cells which have no axis-cylinder processès. The instance—the cells of Clarke's column—from which he endeavoured to prove that certain cells have no axis-cylinder processes, was unfortunate, since it has been shown, by the same methods which Gerlach himself used, that he was mistaken. However, the existence or non-existence of cells without axis-cylinder processes is not essential, and, for purposes of comparison, it is better to suppose that they do not exist.

The Gerlachian theory, then, stands thus: the grey matter is a network; long fibres of every degree, whether destined for other parts of the homologous network or for a heterologous network (say from the grey matter of the spinal cord to the cortex cerebri) or for a motor organ, start from cells; fibres from distant cells end by ramifications; there is no functional difference between protoplasmic and axis-cylinder processes. It is very important that this last point should be understood. A good deal has been made lately of the difference in direction of conduction. Ramón y Cajal has formulated a theory, “de la polarisation dynamique des éléments nerveux;” v. Gehuchten had already given expression to the same idea in coining the two terms “cellulipetal” and “cellulifugal”; but this difference in the direction of conduction, if it be absolute, which is open to question, is a trivial and accidental distinction. The function of a nerve fibril is to conduct. We should not expect to find a difference in structure or in behaviour to reagents between fibrils which conduct towards a cell and fibres which conduct away from a cell. The two processes of a cell of a root-ganglion are of equal standing; not one “protoplasmic” and the other “neural.”

The essential difference between Gerlach's teaching and the modern view (the view of Retzius, Ramón y Cajal, Kölliker, v. Gehuchten, and many others) turns upon this:—*Is the grey matter a network of continuous strands or is it a felt-work of interlacing but discontinuous fibrils?*

The importance of this distinction lies in its physiological application. If grey matter is a network, impulses are conducted through continuous circuits. If it is not a

network, their passage is an *actio in distans* (cf. Kölliker, *loc. cit.*, p. 35). The substitution of the term "contact" for "contiguity" does not help matters physiologically even if it were justifiable; whereas, anatomically, it is a gratuitous assumption. It is impossible for a microscopist to distinguish, in a preparation stained black, between *contact* and *continuity* of substance. Besides he has no right to conclude that the position of structures in a hardened and shrunk tissue is the position which they occupy during life. On the contrary, he knows that the tissue has lost lymph to the amount of at least one-third of its bulk.

The extreme difficulty of this conception justifies us in pushing our resistance to the verge of rebellion. I wish, therefore, to lay before you certain considerations which seem to justify us in calling for yet more evidence before we make up our minds.

(1) The belief in the discontinuity of the elements is based chiefly, although not entirely,¹ upon results obtained with the chrome-silver method. Certainly the figures which this method gives appear discrete; but what is proved by a stain which picks out one cell-system and leaves a number of similar cells uncoloured? In no preparation of the cortex cerebri, for example, could I find fewer than seven pyramids unstained for every one stained; usually the proportion of unstained cells to stained cells is much greater. Supposing for a moment that all these pyramids are united together by their protoplasmic processes, should we recognise it in a preparation in which every stained cell is surrounded by seven cells which are unstained?

Gerlach's theory, however, did not imply that *homologous elements* are united together. It is more intelligible on the supposition that the large cells (the nutrient cells of long fibres) are not directly connected with one another. Now when we endeavour to obtain evidence as to the union of heterologous elements we find that the Golgi preparations are almost silent. Hardly ever are two distinct kinds of nerve element, say branching fibres and nerve-cells, or large

¹ Forel in 1887, Retzius (using Ehrlich's method), and His (as the result of embryological research) were led to doubt the existence of the network.

nerve-cells and granules, properly and completely stained in the same part of any preparation, or even in the same preparation.

(2) Even with the chrome-silver method a coarse union between similar cells is shown to be by no means uncommon. These connections appear to be freaks in development, and are of no interest except as showing that intercellular union is possible. In fig. 8 I show a case in which the apical process of one pyramid ran into the base of another; careful focussing left no doubt as to their actual continuity. Union between two cells of Purkinje by means of a thick arched process is not uncommon. Fig. 9 shows three cells of the spinal cord connected by stout protoplasmic processes. I have seen two granules in the olfactory bulb of an adult animal united by a stout bridge.

(3) *Thorns* (épines) on the protoplasmic processes of pyramids and cells of Purkinje were first described by Ramón y Cajal, but no attempt has been made to determine their significance. I am inclined to think that these thorns will eventually give the key to the problem.

Thorns are very minute lateral branches of the protoplasmic processes of certain cells; especially pyramids (figs. 2, 11), and cells of Purkinje. They often occur at very regular intervals. Sometimes they are opposite but usually they are roughly alternate. Their length varies from less than 1μ up to 5 or 6μ ; on the peripheral processes of the granules of the olfactory bulb they often measure 10 or 12μ (fig. 18). They usually terminate in minute knobs. Sometimes the knobs alone are visible. Occasionally a clear line can be recognised carrying a round dot in its centre or at one end.

Are the thorns artifacts? They certainly are most distinct in imperfectly hardened tissue. When I first succeeded in making a thoroughly successful injection of the brain with bichromate of potassium I was much struck with the fact that the processes of the pyramidal and other cells were perfectly clean cut and destitute of thorns (fig. 10), whereas tissue which had been purposely left for twenty-four hours before immersion in the osmio-bichromate mixture showed the thorns in a very marked manner (fig. 11). So, too, did

the brain injected with strong salt-solution. This led me to think that the thorns were artificial products. On the other hand, their great regularity is opposed to an artificial origin, and besides, they are not limited to preparations made with the chrome-silver method. If a section of the molecular layer of the cerebellum is deeply stained with hæmatoxyl in and then broken up with needles in glycerine, the thorns on the processes of the Purkinje-cells are beautifully distinct, although they are irregular in length and do not end in knobs.

It seemed to me that the thorns must indicate an actual union between certain unstained filaments and the protoplasmic processes. I sought, therefore, to ascertain the nature of these unknown filaments by studying the cortex of the cerebellum. As everyone knows, the molecular layer of the cerebellum exhibits a very regular striation parallel with the surface. This is seen to perfection in the cerebellum of a cartilaginous fish. In the skate, for example, the undulating processes of the cells of Purkinje, which have a radial arrangement, cross the tangential striæ at right angles, making with them a diaper pattern of the utmost regularity. Near the dorsal commissure, the cells of Purkinje are absent, and only the tangential striæ are present. In the cerebellum of the skate (fig. 12), coloured with the chrome-silver method, I am able to show in place of thorns on the Purkinje-cells, long striæ streaming away from the protoplasmic process on either side. Unfortunately, I have not succeeded in following the axis-cylinder processes of the granules of the fish's cerebellum from the granules to their divarication in the molecular layer, nor had Fusari¹ better fortune. Schaper² traced the fibre into the molecular layer, and observed its divarication, but could not follow it to its termination. We may therefore conclude that the arrangement of the axis-cylinder is the same as in birds and mammals, and I think that there is little doubt but that the tangential fibres which I observe in direct continuity with

¹ Fusari, "Untersuchungen über die feinere Anatomie des Gehirnes der Teleostier," *Int. Monatschr.*, 1887.

² Schaper, "Zur feineren Anatomie des Kleinhirns der Teleostier," *Anat. Anzeiger*, Nos. 21 and 22, 1893.

the protoplasmic processes of the Purkinje-cells, are the axis-cylinder processes of granules.

It is most probable that the tangential striæ seen in carmine or hæmatoxylin stained sections of the cerebellum are the tangential fibres to which the axis-cylinder processes of the "granules" give rise, by their division, in the form of a T or a Y. This T-division was first described by Ramón y Cajal, and has been observed in a large number of animals by Pedro Ramón. In many of my own preparations, the delicate axis-cylinder is to be seen commencing in the granule, passing between the cells of Purkinje, and divaricating in the molecular layer. It is important to notice that the branching system of Purkinje-cells, and of the "molecular" cells are arranged at right angles to the folia, the longitudinal fibrils in the direction of their long axes.

This brings us back to the old view as to the plan of structure of the cortex cerebelli, first defined by Boll and often reiterated by myself and many others. Afferent fibres branch among the granules; delicate processes from the granules stream into the molecular layer between the cells of Purkinje; dividing at right angles (as demonstrated by Cajal), they are collected by the protoplasmic processes of the Purkinje-cells, from the base of each of which a neural process passes into the medullary centre. This seems to be the basis of the reflex machine—the system of primary couples—to which the bracket-like cells of the molecular layer, the collaterals of the neural processes of the cells of Purkinje, the cells of Golgi, the fusiform cells, the scattered large cells of the granular layer, and other elements, must be fitted in as our knowledge of the connections of these elements becomes more complete.

If in the cortex cerebelli a *direct continuity of structure is established from the granules to the cells of Purkinje*, a similar continuity must be looked for in the cortex cerebri, and may be expected in the central grey tube.

The fact that the silver deposit extends so short a distance along the tangential fibrils as to give to the protoplasmic process merely a "thorny" appearance, seems to

indicate that GREY MATTER CONSISTS OF TWO SUBSTANCES WHICH BEHAVE DIFFERENTLY TO THE CHROME- OR SILVER-SALT. The ending of the thorn in a knob, as well as the frequent varicosities in the course of all other delicate fibres, especially at the points at which collaterals are given off from axis-cylinder processes, indicates that the substance which reduces the silver-salt is fluid or semi-fluid. The reduction of the silver by neurogleial cells, and the frequent appearance of the deposit outside nerve cells, show, I think, that it is *not the nerve strand properly so-called, i.e., the conducting fibril,¹ which reacts to silver, but the fluid or semi-fluid substance—the neuroplasm—in which it is embedded.* If this hypothesis be correct, it may be applied in many ways to explain the fact that a direct continuity of heterologous nerve elements has not been observed (except by myself in the case of the cerebellum) with Golgi's method.

The chief difficulty which I see in the way of this explanation is the fact that many ependymal cells are quite as thorny as the protoplasmic processes; but this difficulty has to be faced whatever view is held as to the meaning of the thorns. No doubt the ependymal cells are constructed upon the same plan as the web which they serve to support.

It will, no doubt, be urged in opposition to my theory, that it is not only Golgi's method which seems to demonstrate the discontinuity of nerve elements; the same thing is exhibited by Ehrlich's method, which has given such admirable results in the hands of Retzius; but I maintain that this fact is confirmatory of my hypothesis. Methyl blue injected *intra vitam* acts very quickly upon the nerves, while they are in a living state. It is much more likely that the stain is taken up from the circulation by the nutritional protoplasm of the nerves than that it affects the conducting fibrils.

The fact that NO THORNS ARE TO BE SEEN WHEN THE BRAIN IS INJECTED WITH BICHROMATE shows that the outward spread of the neuroplasm along the nerve-fibrils occurs *post-mortem*. We should, therefore, expect that Ehrlich's

¹ The question as to whether the fibrils are solid (Schultze) or hollow tubules (Nansen, Schäfer) does not affect the theory.

method would show the nerve-elements as still more discrete than they seem to be when prepared in the ordinary way by the chrome-silver method after death.

Lastly, I submit that even from the point of view of those who work chiefly with Golgi's method, it is a bold assumption that this method shows us the ultimate termination of nerve-fibrils. Speaking for myself I may say that the thought which a good Golgi-preparation of the spinal cord suggests is not, how complete the picture, but how much there must be that one does not see! The amazing delicacy of the last divisions of the collaterals impresses upon one's mind the fact that it is hopeless to try to follow them to their termination since the method is incapable of showing their ultimate subdivisions.

Still more forcibly is the inadequacy of the method brought out by a study of the cortex cerebri. Every here and there (especially when the tissue has remained a long time—say six weeks—in bichromate and osmic) occur masses of fibrils—looking like the mycelium of a fungus. The centre of the mass is an impenetrable tangle, and only at its margin can the separate fibrils be seen; it brings home to one the fact that admirable and diagrammatic as are the partial pictures, they give a very incomplete and inadequate representation of the infinitely complex tissue of the brain.

Granules.—Perhaps the greatest service which Golgi's method has rendered to histology is in giving us accurate information with regard to the nature and form of these minute nerve cells. All attempts to isolate the granules by teasing had failed; fragments only of their processes remained attached to the cell-body. Owing to the closeness with which they are massed together there was no hope of our being able to make out their form by studying them in sections. The chrome-silver method came to our rescue; picked out one granule in a hundred and depicted it with diagrammatic simplicity.

Since our knowledge of the granules commences with the introduction of Golgi's method, it is superfluous to quote the views of observers who had not this means of observation at their disposal. Granules were called myelocytes, connec-

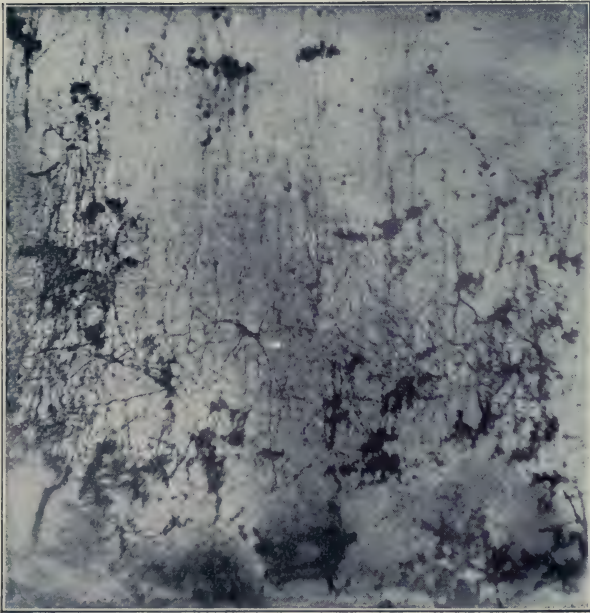


FIG. 17

Adult cat ; olfactory bulb. At the bottom of the photograph are seen the glomeruli. The mitral cells are very imperfectly coloured. Above the mitrals is seen a single cell of the type for which I suggest the name "bracket-cells." Its protoplasmic processes extend horizontally for more than 3 mm. Its axis-cylinder was centripetal. Numerous granules are seen above this.

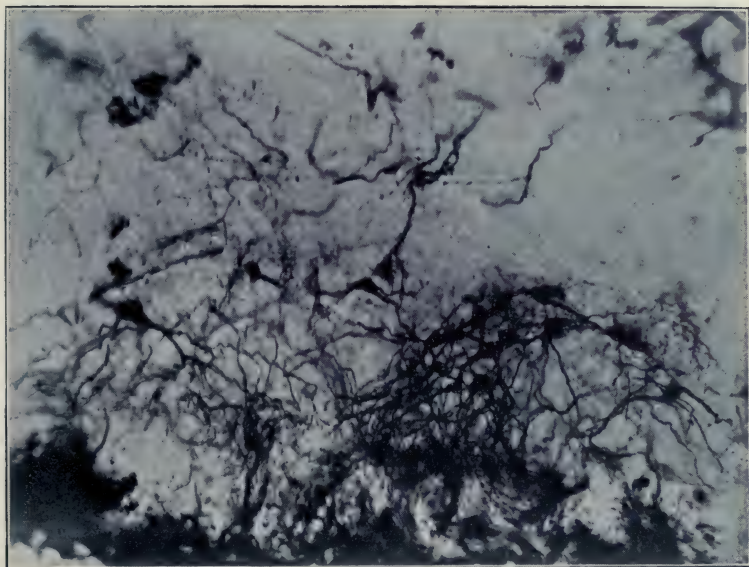


FIG. 18.

Kitten, 3 days old; olfactory bulb. Showing mitral cells and the extremely thorny protoplasmic processes of the granules.



FIG. 19

Olfactory bulb ; granules with axis-cylinders selected from various preparations :

1-4. Hedgehog ($9\ \mu$ to $11\ \mu$).

5. Kitten, 3 days old ($10\ \mu$).

6, 7. Rat, 12 days old ($12\ \mu$ and $15\ \mu$).

By an oversight 1, 2, 3 and 4 are drawn with their central ends upwards ; 5, 6 and 7 with their central ends downwards.

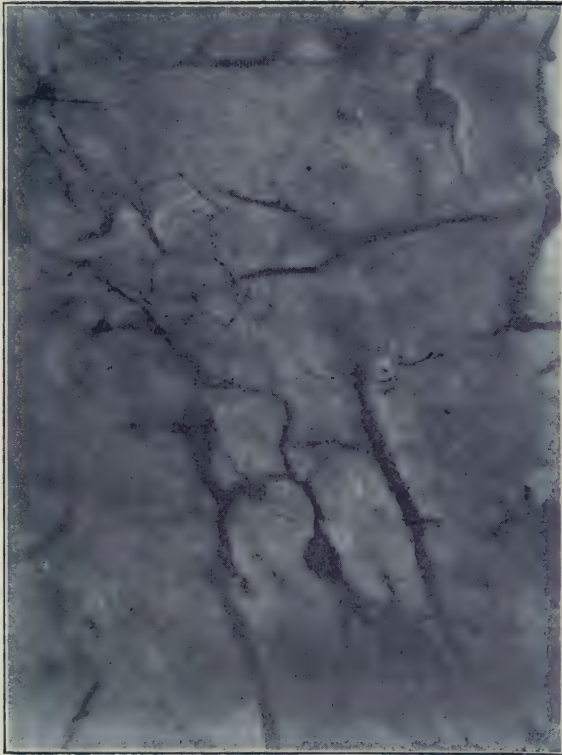


FIG. 20

Photograph of the granule 19, 7 (reversed).

tive tissue corpuscles, lymph-corpuscles, "doubtfully nervous" elements, and so forth, by men who would be the first to acknowledge that their nervous nature is now placed beyond any possibility of doubt. It is a little strange to find the hypothesis put forward in a recent memoir that they are nerve-cells which have remained behind in an embryonic condition.¹

There are four situations in which granules are found in dense masses: (1) the olfactory bulb; (2) the retina; (3) ? the fascia dentata; (4) the cerebellum. They are very numerous in the occipital region of the cortex cerebri. That all these small nerve cells bear a similar relation to afferent fibres is not improbable; but whether on this account they should all be described as homologous is a question which cannot be answered until we have further information. In the cerebellum the granules are arranged about clumps of molecular substance (figs. 3 and 13). So far as we can see, they are all of one kind, but Denissenko² thought that he could distinguish a second type of nucleus by the use of eosin. I have never been able to find these eosinophile nuclei of Denissenko, although I see in the centre of each clump of molecular substance the structures which he mistook, as I think, for nuclei. These structures are usually smaller than the nuclei, homogeneous and pale; sometimes single, and round, they often appear as rods, and very frequently they are divided into several parts (fig. 3).

When I first observed these structures in sections stained with eosin, or better still with the alum-carmine-Weigert

¹ Chatin, "Études d'histologie Zoologique sur la forme dite Myélocyte," Paris, 1890.

I am not quite sure that I have given a fair representation of Chatin's views. The following quotations show that Chatin looks upon the granules as identical with the large nerve cells when in an embryonic stage; but he does not state explicitly that either the granules are capable of growing into larger cells, or that they are larger cells which have remained in an embryonic, and therefore functionless, condition. "Ce qui mérite d'être noté, c'est l'identité morphologique qui se manifeste constamment entre tous ces jeunes éléments nerveux, quelle que soit l'espèce animale sur laquelle on en suit l'évolution." "Il ne suffit pas d'avoir montré l'exacte signification du myélocyte, il faut encore établir qu'il ne peut aucunement représenter un type distinct."

² "Zur Frage über den Bau der Kleinhirnrinde bei verschiedenen Klassen von Wirbelthieren," *Archiv. für Mikr. Anat.*, Bd. xiv., 1877, p. 203.

method, I concluded that they were not nuclei, but did not like to hazard the suggestion that they were, as I thought them to be, swollen axis-cylinder processes at their points of division. The use of the chrome-silver method shows, however, that this is really the case. They are the dilated fibres of the rosettes which occur on the course of Ramón y Cajal's mossy fibres (*fibras musgosas*).

The granules arranged around each molecular clump give protoplasmic processes to it (as well as to three, four, five, or even six other clumps). Their protoplasmic processes end in curious bent claws which are in relation with the processes of the rosette. Whether this connection is one of contact only or of direct continuity remains to be proved.

If we neglect for a moment the nuclei which have been supposed to occur within the molecular clumps, and eliminate, of course, the nuclei of the superficial "Golgi cells" and the scattered large cells, we have to deal with a vast number of apparently similar nuclei. The question arises: Are all these of one kind? The way in which the chrome-silver method picks one granule out of several hundred, is more remarkable even than its capricious selection of other kinds of cells; I have never seen the granules of the cerebellum stained in masses, as often occurs in the olfactory bulb. They are always disposed at fairly regular intervals. All observers who have worked hitherto with the chrome-silver method have concluded that only the type of granule with centrifugal axis-cylinder is present. I shall show in a moment that granules of quite different form with centripetal axis-cylinders are also to be found.

In addition to its short protoplasmic processes already described, each granule (of type 1) has a single axis-cylinder process of exquisite delicacy which makes its way into the molecular layer and ends, as Ramón y Cajal discovered, by dividing at right angles to the planes of the Purkinje-systems.

The enormous number of radial fibrils which in some sections are seen making their way to the molecular layer between the cells of Purkinje as well as the equally numerous tangential fibres found in the molecular layer in other

sections, evidently require a vast number of granules to give them origin.

GRANULES WITH CENTRIPETAL AXIS-CYLINDERS.

It is late in the day to describe a new kind of cell in the cerebellum, but it is a curious proof of the caprice of the chrome-silver method that these granules should not have shown themselves to other observers. In one set of preparations from a four-day rat, they occur in large clusters about the apices of the convolutions. I have not yet had time to look for them in other subjects.

These new granules are quite unlike any hitherto described, nor can they be confused with the fusiform cells described by Golgi. Golgi's cells are much larger ($20\ \mu$), with long protoplasmic processes from each end of the spindle, and a fibre attached to the side of the cell. The new granules have a transverse diameter of $9\text{--}12\ \mu$. From the apex, a branching protoplasmic process runs towards the periphery (figs. 14, 15), while an axis-cylinder process is attached to the base. The axis-cylinder frequently gives off a recurrent branch near the granule, and passes on towards the white matter where or near which it divides.

What are the *central connections of the granules*?

From the medullary substance of the *arbor vitæ* a single medullated nerve fibre traverses the granular layer to reach each Purkinje cell; but there can be no doubt that many fibres are left over after all the Purkinje cells are provided for. Amongst these are the mossy or rosette fibres, which form an extremely complicated and delicate branching system among the granules. In animals in which the medullary fibres separate the granular layer from the cells of Purkinje, some fibres turn towards the molecular layer, whilst others turn towards the granules. This is seen especially well in cartilaginous fishes and in some reptiles, for example, the crocodile. The fibres which turn towards the granules divide frequently, and it is on this account, probably, that there is a marked difference in size between the fibres near the cells of Purkinje and those near the granules. The former are much the larger.

Ramón y Cajal has assigned to the granules with centrifugal fibres their proper position as elements on the ascending limbs of the cerebellar reflex arcs.

The *granules of the olfactory bulb* have not been treated with so much respect as those of the cerebellum. Many considerations show us that the granules of the bulb cannot be regarded as strictly homologous with the granules of the cortex cerebelli or cortex cerebri. The organ in which they are found is widely dissimilar from the cortex, homologous with a peripheral nervous organ—the retina—and not with a central or secondary nerve field.

Nevertheless, the granules of the olfactory bulb (figs. 16, 17, 18, 19) have sufficient in common with the granules of the cerebellum to entitle them to the full status and privileges of nerve cells. To simplify the diagram of the olfactory bulb by leaving out the granules, as v. Gehuchten has done,¹ is to complicate matters for future generations of histologists:—
“Les cellules mitrales sont donc les véritables cellules olfactives du bulbe. Loin d'être, comme Golgi le croit, une voie de transmission accessoire, elles forment l'unique route par laquelle l'ébranlement nerveux d'une fibrille olfactive peut être sûrement conduit jusqu'au cerveau.”²
This seems a little hard upon the granules, not to mention various other kinds of nerve-cell, which together play a much larger part in the formation of the olfactory bulb than do the mitral cells!

The granules of the olfactory bulb usually appear as minute round, fusiform or triangular cells with one very long peripheral process, which passes through the layer of mitral cells, and often approaches very near to the glomeruli, and three or four much more delicate central or basal processes which are often coloured only for a short distance. Occasionally the central processes are long and branch frequently. Sometimes the round cell-body lies on the side of a process which then extends for a long distance centrally as well as peripherally. It is rather remarkable that the peripheral

¹ Cf. Van Gehuchten, “Système Nerveux de l'homme,” Lierre, 1893, figs. 390-393.

² V. Gehuchten and Martin, *La Cellule*, vii., 1891, p. 232.

process is stained much more easily than the central processes.

Both peripheral and basal processes are thorny (fig. 18); the thorns on the sub-divisions of the peripheral process, after it has passed the layer of mitral cells and entered the stratum gelatinosum of Lockhart Clarke, are remarkably long (6-12 μ).

Golgi is doubtful as to the nervous nature of these cells, although he thought he saw in certain rare cases an axis-cylinder process which gave off collaterals and joined bundles of nerve fibres; as did also its collaterals.¹

Ramón y Cajal failed to find any axis-cylinder processes, but he nevertheless considers that the form of the cells and the termination of their apical processes in close relation with the protoplasmic processes of the mitral cells justify us in considering the granules as nervous. He homologises them with the "spongioblasts" of the retina as amacrine cells—*i.e.*, cells destitute of axis-cylinder processes.

V. Gehuchten and Martin, on the other hand, consider that since the granules have no axis-cylinder processes they are not nervous: "Du moment que, à l'exemple de Ramón y Cajal, on est disposé à ne tenir aucun compte de la présence ou de l'absence d'un prolongement nerveux pour déterminer le nature spéciale d'un élément des centres, on perd par le fait même tout motif pour ne pas considérer comme étant de nature nerveuse non seulement les cellules épendymaires . . . mais aussi un grand nombre de cellules de neuroglie."

I should have thought with Ramón y Cajal that the general appearance and disposition of the granules of the olfactory bulb sufficiently testify to their nervous nature; although the presence of an axis-cylinder may be needed to place it beyond a doubt. For the axis-cylinder I have sought very carefully in a large number of different prepara-

¹ Golgi ("Untersuchungen über den feineren Bau des Centralen und peripherischen Nervensystems"; German translation, Jena, 1894, p. 52) says: "Ob auch ein anderer, nervöser Fortsatz besteht, Kann ich nicht sagen. Allerdings habe ich in einigen Fällen beobachtet, dass einer der Fortsätze, welcher gewöhnlich aus dem Mittleren Theile der Basis der Pyramide entspringt, etwas homogener aussieht, als die anderen"

tions, with the result that I can almost always find it in the case of a certain number of the granules in any given section of the olfactory bulb (fig. 19).

The axis-cylinder is a short curved process attached to the side of the granule. As it leaves the granule it twists among the other granules which are densely packed together, and is soon lost to view. I have failed to determine its further course, although I notice that it inclines towards the periphery, but I would call attention to the fact that the tissue which separates the plates of granules is occupied by a dense feltwork of fibres; some derived from the basal protoplasmic processes of the granules, but the rest distinguished by the extreme delicacy which is characteristic of axis-cylinders and their collaterals. Occasionally the axis-cylinder process divides at right angles, very near the cell, one division running centrally, and the other peripherally.

Solitary granules are to be found at the margin of the special layer of the olfactory bulb which contains mitral cells and glomeruli. Their axis-cylinder processes may be followed for some distance, but I have not succeeded in ascertaining their destination.

Fig. 20 shows the axis-cylinder process of a cell of the olfactory bulb of a twelve-day old rat; but I am not certain whether this cell ought to be regarded as a granule. It lay at some little distance from the sheet of mitral cells, giving protoplasmic processes in each direction; those which approached the mitral cells being richly branched. Its axis-cylinder process *ran peripherally* and could be followed almost up to the mitral cell layer. The cell was about half as large again as the ordinary granules, and yet I think that the disposition of its protoplasmic processes and the course of its axis-cylinder process entitled it to be classed with them. It is worthy of note that the axis-cylinder processes of several other granules were distinctly visible in this preparation, and that they also curved peripherally.

If the cell I have photographed be not a granule it belongs to a kind of cell not described hitherto. It is too small for one of the multipolar cells, often of a fusiform shape, described by Golgi, who says, besides "Der nervose

Fortsatz entspringt Gewöhnlich von der nach dem Centrum gerichteten Seite der Zelle, bisweilen auch seitlich, um sich dann nach innen umzubiegen" (*loc. cit.*).

In passing it may be noticed that I do not find any description of the immensely large "bracket-cells," which are common features, at any rate, in both cat and hedgehog. They lie on the central side of the mitral cells, and their branching processes often extend horizontally for a distance of 2 to 3 mm. (fig. 15).

The possibility of the existence in any situation of amacrine nerve cells is too wide a question for discussion here; but it does not appear to me probable that they are to be found in the olfactory bulb. The observation of Pedro Ramón¹ that the lower the animal in the vertebrate scale the less developed are the basal processes of the granules of the bulb, seems to dispose of the idea that these cells are bipolar in the sense of having protoplasmic processes for both cellulipetal and cellulifugal conduction; it makes the existence of an axis-cylinder process for cellulifugal conduction the more necessary.

As everyone knows, the overlapping plates of granules constitute the most conspicuous feature of the olfactory bulb. It seems to me impossible to doubt that they take a large and important part in the constitution of the bulb as a nervous mechanism. A diagram of the structure of the bulb from which they are omitted is not only inadequate but misleading.

Apart, however, from the question as to whether, in the case of the olfactory bulb, we can or cannot prove that the granules are nervous, I wish to claim for them, on general grounds, a much more important rôle in the nervous mechanism than has been conceded to them by any observer who has up to the present made use of the chrome-silver method.

No one now doubts the nervous nature of the granules of the cerebellum. Yet other granules which, when prepared with carmine, picrocarmine, or any other stain very closely

¹ Ramón y Cajal, "Nouvelles Idées," p. 108.

resemble the granules of the cerebellum, are either set aside as non-nervous, or ignored altogether. It is instructive to compare a carmine-stained section of the cortex cerebri with any of the pictures which have been published of the same tissue prepared with the chrome-silver method. In the chrome-silver preparations the granules are conspicuous by their absence; whereas in a carmine preparation of the occipital cortex, or of the cortex about the calcarine fissure, their importance is beyond dispute. It is greatly to be desired that some one should work out the connections of the granules of the cortex cerebri with the aid of the chrome-silver method.

The Granules of the Fascia Dentata.—I have tried in vain to colour these granules with silver.

It may be doubted whether there are any granules in this layer. Sala¹ omits them altogether when recording the results of his observations with the chrome-silver method; but there seem to me to be strong reasons for thinking that the greater number of the nuclei of the "granular layer" of the fascia dentata belong to granules.

The cells of this layer, when coloured with silver, very closely resemble miniature Purkinje-cells. The body is rounded or oval; from its base an axis-cylinder process passes towards the nucleus fasciæ dentatæ. Its apex gives rise to a rich branching system (fig. 21). Sala figures these cells as placed at very regular intervals, as my preparations also show them. The coloured cells lie almost exclusively on the surface of the granular layer, as if they differed in nature from the "granules" which lie below them. In my description of the fascia dentata prepared with the carmine Weigert stain² I state that the average numerical relation of the small pyramids to the granules is as 1 to 8. This number coincides approximately with the relation between the unstained nuclei and the stained cells in my chrome-silver preparations.

A third reason for considering the cells of the fascia

¹ *Zeitschr. f. w. Zoologie*, vol. lii., 1, p. 18.

² Hill, "The Hippocampus," *Phil. Trans. of the Royal Society of London*, 184, B. 1893, p. 389.

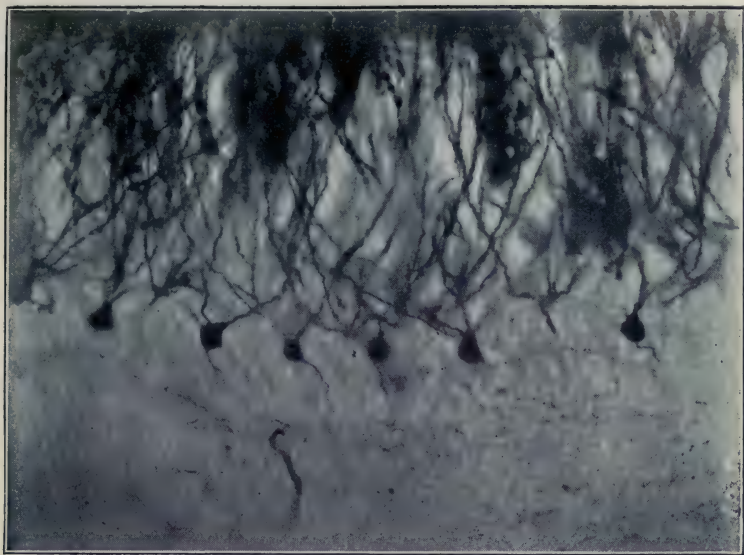


FIG. 21.
Adult hedgehog; fascia dentata hippocampi $\times 310$.



FIG. 22.
Adult hedgehog: subiculum cornu Ammonis. Showing double pyramids.

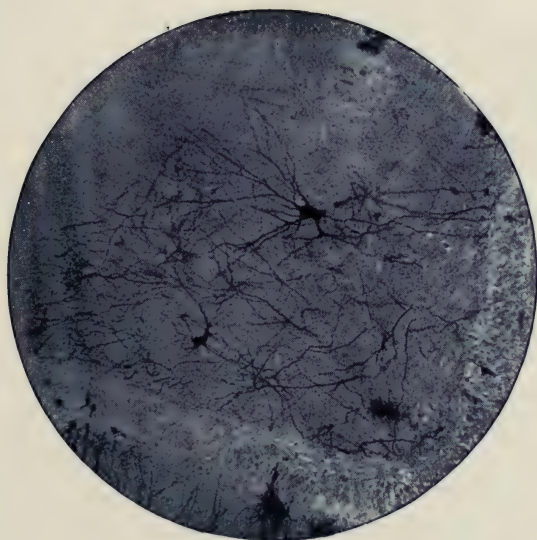


FIG. 23.

Adult hedgehog; nucleus fascial dentata $\times 83$. Showing multi polar cells.



FIG. 24.

Adult hedgehog; spinal cord. Cells of the anterior horn. A single nucleus is also coloured.

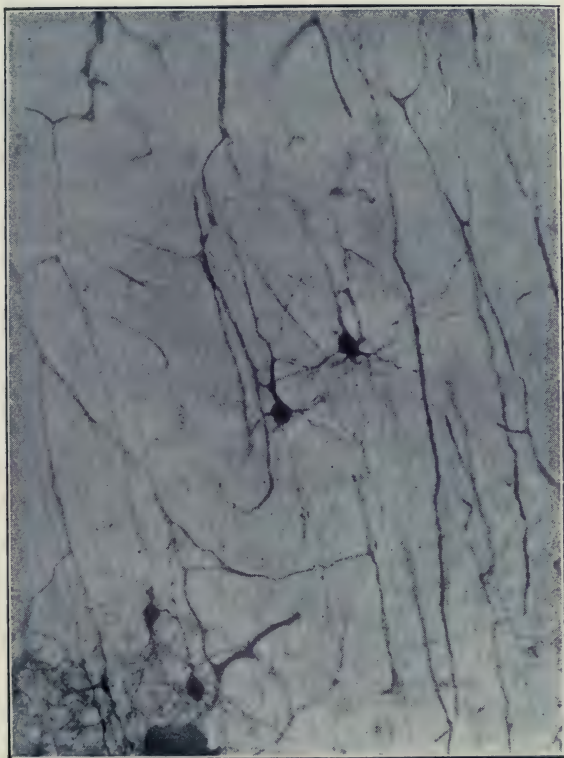


FIG. 25.

Rat, 8 days old; cortex cerebri $\times 155$. The collaterals on the axis-cylinders of two pyramids are well seen.

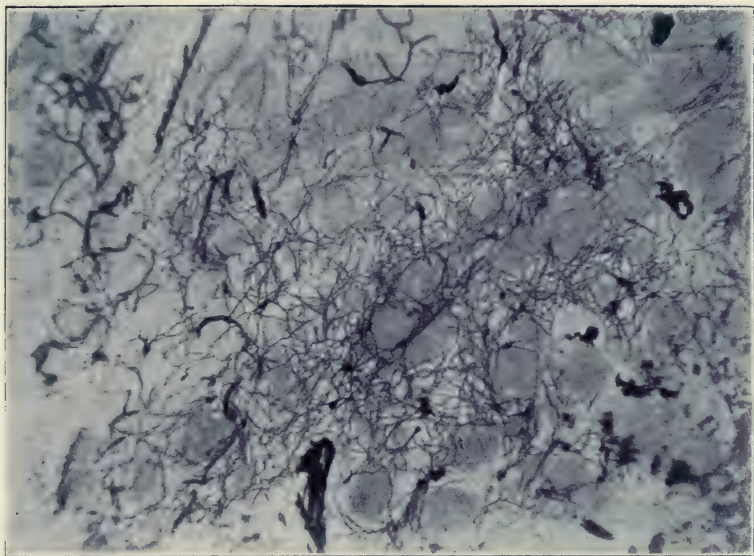


FIG. 26.

Adult hedgehog; nucleus tentacularis. The blood-vessels were injected with warm bichromate of potassium. The reaction shows cells of Golgi's type II., and also very faintly the axis-cylinders of the fibres which constitute the bundles of the internal capsule.

ALL THE PHOTOGRAPHS ARE ABSOLUTELY UNTOUCHED BY HAND.

dentata as granules is also brought out in the paper just alluded to, although its value as a proof is less obvious. The development of the fascia dentata is exactly proportional to the development of the olfactory bulb. It is conceivable that the granules in the two situations belong to the same formation.

It may not be out of place in this description of the fascia dentata to call the attention of histologists to the illustration which the hippocampus affords of the extreme difficulty of basing homologies upon the form and general appearance of nerve-cells. As the hippocampus is approached, the pyramids of the cortex cerebri give place to the remarkable "double" pyramids of the subiculum cornu Ammonis (fig. 22); at the nucleus fascia dentata these are transformed into cells, which might easily be mistaken for the motor cells of the spinal cord (fig. 23); in the fascia dentata (fig. 20) we have a type of cell for which we most easily find an analogy in the cortex cerebelli.

Summary.—The method of colouration introduced by Professor Golgi, of Pavia, has yielded, to those who have had the foresight to apply it persistently, an undreamed of harvest. In particular Santiago, Ramón y Cajal is to be congratulated upon the wealth of new facts which it has enabled him to discover. These are, I believe, only a first instalment of the revelations in nerve-histology which the method is destined to make.

I have used it, not with the hope of making discoveries, but with a view to ascertaining the conditions of its action and the value of its results when compared with those which other methods afford. In other words, I have tried to discover its limitations—its weakness rather than its strength.

The subject is so large, and my results so fragmentary, that I hesitate to lay them before you, and I should not, assuredly, have embodied them in their present incomplete form in a memoir. When, however, you did me the great compliment of electing me as your President, I felt that you would expect me to bring before you such results of my personal work as are likely to be of greatest interest to neurologists in general.

The chrome-silver method has completely transformed our conception of the nature of the nervous mechanism. Whatever branch of neurology we may happen to pursue, we need to lay afresh the foundation upon which it rests. The new method of colouration has revealed to us the fact that grey matter is far more complex in its structure than histologists imagined. We are also asked to throw aside all preconceived ideas as to its mode of working, and to believe that its various elements are not in structural continuity. It is on this latter point that I wish you to suspend your judgment, and in favour of my *caveat* I lodge the following considerations:—

(1) The place of the granules in the mechanism has not yet been ascertained except in the case of the cerebellum. In all cortical fields they are in intimate relation with the bulk of the ascending or afferent fibres. How close their connections with these fibres may be, still remains to be determined. Their axis-cylinder processes are also in intimate relation with the protoplasmic processes of the cells which give origin to efferent fibres.

(2) The thorns on the protoplasmic processes of nerve cells are not artifacts, nor are they, on the other hand, processes which the chrome-silver method reveals in their entirety. It is not impossible that they contain the ends of the axis-cylinder processes of the granules surrounded by an extension, or *post-mortem* overflow of the neuroplasm of the larger nerve cell.

If a direct continuity of nerve substance between axis-cylinder processes of granules and the protoplasmic processes of nerve cells be proved to exist, it may be looked for also along all other lines of impulse reflection, and Gerlach's plexus—with all the modifications in form introduced by the chrome-silver method—may be a true expression of the structure of grey matter as a nervous mechanism after all.

ON THE INFLUENCE OF REAGENTS ON THE ELECTRICAL EXCITABILITY OF ISOLATED NERVE.

BY AUGUSTUS D. WALLER, M.D., F.R.S.

Syllabus.

Introductory Remarks.

Demonstration of Representative Experiments.

Action of Gases.—Oxygen—Carbon Dioxide—Carbon Monoxide Hydrogen—Nitrous Oxide—Hydrogen Peroxide—Chloroform—Ether—Hydrocyanic Acid—Amyl Nitrite—Ammonia—Chlorine—Coal Gas.

Influence of Temperature.—Decline and Reversal of the Currents of Injury and of Action—Effects of Alcohol and of Tobacco Smoke.

Action of Reagents in Solution.—Conditions of Observation—Bromide of Potassium—Chloride of Sodium—Water—Acids and Alkalies.

Alkaloids.—Muscarin, its Case Considered in Detail with Reference to the General Method of Procedure—Nicotin—Curarin—Atropin—Cocain—Pilocarpin—Strychnin—Aconitin—Veratrin—Gelseminum—Chloral Hydrate—Butylchloral Hydrate—Antipyrin.

Comparative Action of Haloid Salts in Equi-molecular Solutions (M_{10})—Bromides—Chlorides and Iodides of Ammonium, Sodium and Potassium—Determination of the Predominant Partner in the Molecule—Action of Calcium, Barium and Strontium—of Rubidium, Cæsium, Gold, Lithium, Copper and Zinc.

Antagonism, Successive and Simultaneous; False and True.

Chloroform versus Ether Anæsthesia.

Comparative Action on Nerve of some Ethers and Ethereal Salts ($[C_2H_5]_2O$; C_2H_5Cl ; C_2H_5Br ; C_2H_5I);

Of Chloromethanes (CH_3Cl ; CH_2Cl_2 ; $CHCl_3$; CCl_4);

And of Chlorethanes (C_2H_5Cl ; CH_2Cl-CH_2Cl ; CH_3-CHCl_2 ; $CH_2Cl-CHCl_2$; CH_3-CCl_3 ; $CH_2Cl-CCl_3$; $CHCl_2-CHCl_2$; $CHCl_2-CCl_3$).

The experiments on the subject of the lecture, and the lecture itself have been considerably extended during the past year, and the portion now published relates mainly to the action of anæsthetics. The accompanying illustrations, in several instances reproduced from records taken since July

6, refer only to the action of CO_2 , chloroform and ether. Evidence relating to other substances mentioned in the text, and to the antagonism of chloroform by CO_2 will be presented in subsequent papers. The three principal experiments described below (CO_2 , abolition then augmentation; Et_2O , temporary abolition then recovery; CHCl_3 , permanent abolition) were demonstrated at the November meeting of the Physiological Society (Proceedings of November 9 in *Journal of Physiology*, vol. xviii., p. 45).

THE first thought that suggests itself when one has seen what a surprisingly regular series of effects is obtained from nerve by regular excitation at regular intervals, is that we are in possession of an admirable test by which to recognise the action of drugs upon the excitability of nerve, and, as we shall presently see, the surprising regularity of the normal effects is paralleled by an even more surprising regularity in the modifications of these effects, caused by drugs.

A priori we should not have anticipated such clear results as we shall witness; nerve, the conducting organ, is, in relation to knowledge of this order, practically a blank sheet; nerve poisons are regarded as acting only upon the central and peripheral end-organs, not upon the conductors, and with the exception of observations by Biedermann, on the effect of ether upon physiological electrotonus, no positive data exist touching this fundamental subject. As far as concerns experimental pharmacology, nerve, apart from its end-organs, does not yet exist.¹

Yet *a priori* again, while fully admitting the superior susceptibility of terminal grey matter to the modifying action of drugs, there would seem to be every probability in favour of our finding the conducting grey matter, *i.e.*, the axis-cylinder, similarly susceptible, if in less degree.

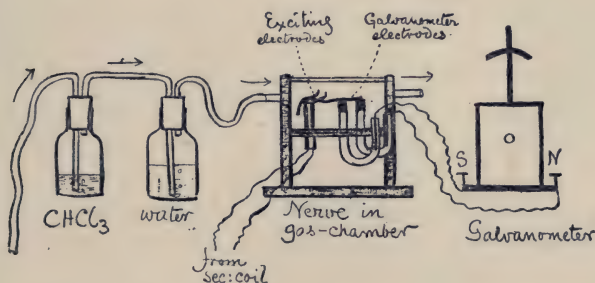
But it is not to speculations alone that I am about to call your attention. I intend to show you that pure nerve is accessible to such experiments—that it is in fact the

¹ I do not, of course, ignore the numerous investigations upon nerve by Ranke, Kühne, Grützner and many others, in which the action of reagents upon nerve has been gauged by muscular and by sensory reactions. The statement made above refers to *isolated* nerve. The only observations relating to isolated nerve that I have been able to find in toxicological literature are those of Mommsen on the action of poisons (*Virchow's Archiv*, 1881, p. 243), inclusive of alcohol, chloroform and ether. Mommsen employed the usual nerve-muscle preparation, but also as a confirmatory test the galvanometer, thus employing nerves that were *isolated* from their end organs.

tissue above all others suited to exact investigation of the chemical action of reagents upon excitable, that is to say, living matter.

Let us begin with gases, with, say, carbon dioxide, chloroform, ether, hydrocyanic acid, &c., preferring these to drugs in solution for the obvious reason that we can most easily apply them with least disturbance to the nerve.

The disposition of things is as shown in this diagram, the parts of which need merely be named in order that their purpose may be understood. A gas-chamber with



To test action of gases on nerve.

FIG. 2.

inlet and outlet, containing a frog's sciatic nerve¹ laid upon exciting and leading-off electrodes, wires from an induction

¹ Mammalian nerve is unsuitable. As was shown at a previous lecture, the negative variation vanishes rapidly from excised nerve. The statement of Hermann as to the long continuance of excitatory phenomena on rabbit's nerve *post-mortem* is correct if by excitatory phenomena electrotonic changes are signified; that of Fredericq as to the existence of a true negative variation in mammalian nerve twenty-four hours *post-mortem*, is, I think, open to question. A true negative variation—i.e., one independent of distance between exciting and leading-off electrodes, and not reversed by reversal of the excitation—has never come under my observations of excised mammalian nerves; electrotonic effects have been particularly prominent; but to what extent such effects are to be admitted as physiological phenomena is an unsettled question that cannot be profitably discussed in this connection. The effects under observation are, to the best of my knowledge, true negative variations, and if to any extent an electrotonic factor has also been present, it has been a physiological phenomenon subject to the influence of excitatory and depressant drugs.

coil to the nerve, and from the nerve to the galvanograph. Two wash-bottles, one for the drug, the next for water, to avoid drying in the gas-chamber when gas is blown or sucked through it.

A couple of representative experiments carried through under your eyes, and, while the experiments are going on, the exhibition of the autographed results of other similar experiments, cannot fail to convince you that we are here in possession of a method of investigation second to no other.

For the test is applied to nerve and to nerve only. There is not, as when muscular contraction is used as the indicator, any question as to the share borne by muscle or by the motor end plate in any effect that may be produced. The isolation of the object of experiment is complete. It does not move. Mere excitation does not exhaust its excitability.

The galvanometer spot on the screen has moved southwards four times during the last four minutes, at each minute the nerve in the gas-chamber has been excited for one-eighth of a minute by completion of a tetanising current of a strength of ten units; as you saw, the successive deflections were of regular magnitude. I now blow chloroform vapour through the gas-chamber, and watch for the next excitation by closure of circuit at the mercury pool. There is no effect, nor at any succeeding closure. The excitatory effect has been abolished by chloroform while I was speaking; the nerve is completely anæsthetised. Its excitability may not, however, have finally disappeared, the nerve is not necessarily dead, for if I disconnect the chloroform bottle and blow moist air through the gas-chamber you may (or you may not) see, if you will wait, a recovery of the effects of excitation. But the reappearance, if it occurs at all, will take some little time, which we may employ upon another similar experiment.

Here is a second gas-chamber, and nerve similar to the first, upon which we will make a double-barrelled experiment, first CO_2 then ether, neither of which will miss fire.

The excitatory effects are as you see quite regular, between each two effects I have turned a reverser so as to reverse the direction of the exciting current in the nerve and the deflection due to the "action current" has remained unaltered. CO_2 gas is now passed through the gas-chamber for about a minute. During the next minute or two the deflections are suppressed, but very soon the suppression (or it might have been only diminution) makes way for an augmentation which might persist for ten, twenty or thirty minutes. But we shall not wait for this.

Ether vapour (Et_2O) is now passed through the gas-chamber for about a minute as before. The deflections that would otherwise have made their appearance each minute are, at any rate after the first minute, completely suppressed. They will, however, soon reappear, even though I do not as in the case of chloroform, air the chamber, they will do so in a few minutes and they may (it is not likely in this case that they will) be progressively increased to a magnitude considerably above the normal.

I do not wish to smother these main facts with details, however important the latter may seem to be, but before stopping these experiments, will prolong them for five minutes and give you in a few words the results of over a hundred similar experiments with the three reagents, CHCl_3 , Et_2O and CO_2 .

Always the immediate effect of any of these three reagents, when strong, is an abolition of excitability, temporary in the case of CO_2 and ether, often final in the case of chloroform. With CO_2 and ether the nerve comes to without aeration of the gas chamber; I have never seen it do so with chloroform, and have often failed to bring it round by aeration. The first two reagents have a true anæsthetic, *i.e.*, temporary effect, the third is more prominently toxic, and its effect is apt to be final. The after-effect of both CO_2 and ether administered in abundance to the nerve is an augmentation of excitability, most prominent in the case of CO_2 , of which, moreover, an anæsthetic period is briefer than in the case of ether.

Administered in a diluted state, *i.e.*, with much air, all

three reagents give as their immediate effect on nerve an augmentation of excitability that gradually wears off, most rapidly in the case of chloroform, of which the action on nerve is most manifestly toxic.

If time were unlimited, I might go on to show you a series of experiments upon a series of nerves put up in moist chambers, but it will be more economical, while affording you a more comprehensive survey of quietly conducted experiments, if I project their autographed records upon the screen. From the tests and precautions that you have witnessed on previous occasions, you are more or less assured that fallacies have been guarded against, and that there is no question of current escape, or of electrotonic effects, or of unipolar action, or of magnetic action of the induction coil. Moreover, the records will speak for themselves. Here are records of the effect of carbon dioxide, of carbon monoxide, of ammonia, of hydrocyanic acid, of oxygen, of ordinary coal gas, of nitrite of amyl, of hydrogen peroxide, of nitrous oxide—in short, of everything that happened to be within easy reach.

Carbon dioxide we have just studied. Carbon monoxide does nothing. Oxygen does nothing, or very little. Strong ammonia kills, weak ammonia excites. Hydrocyanic acid kills; here are two records (plates 238, 341), the first of HCN vapour as given off to the gas-chamber by blowing into it through Scheele's preparation (? 5 per cent.), the second of alcohol vapour by blowing through absolute alcohol; you could hardly distinguish them from each other. Chlorine from BP water kills; chlorine from BP water diluted twenty times, excites. Hydrogen peroxide excites. Methane excites. Hydrogen sulphide excites, then kills. Nitrous oxide does nothing; anæsthesia by laughing gas is probably anæsthesia by CO_2 .

The effect of temperature upon nerve is equally easily tested. Here, again, we have the satisfaction of knowing that nerve and nerve only is submitted to observation. All that is necessary is to enclose our gas-chamber, nerve and electrodes in a larger box, of which the temperature can be raised and lowered at will. Such an experiment is, indeed,

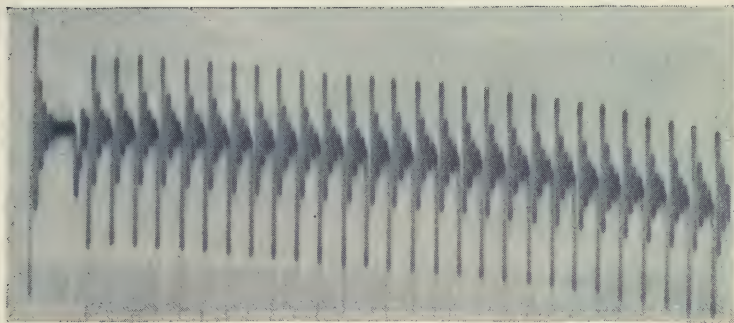


FIG. 1.

A normal series of negative variations.

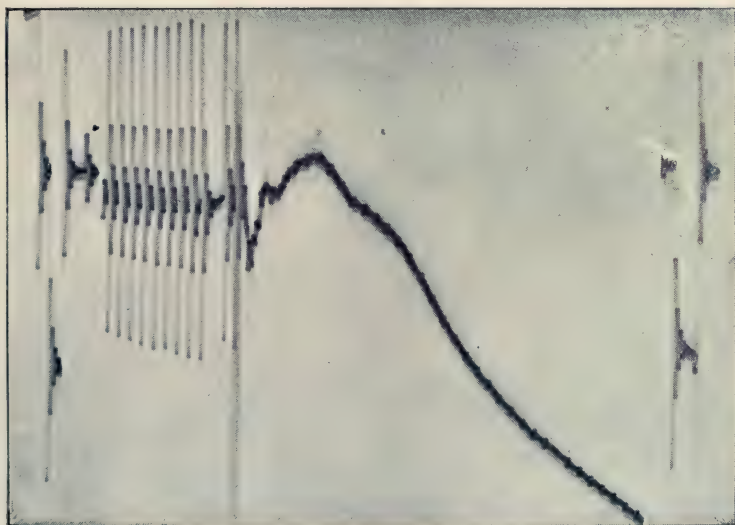


FIG. 3.

Effect of chloroform on the negative variation. Permanent abolition.



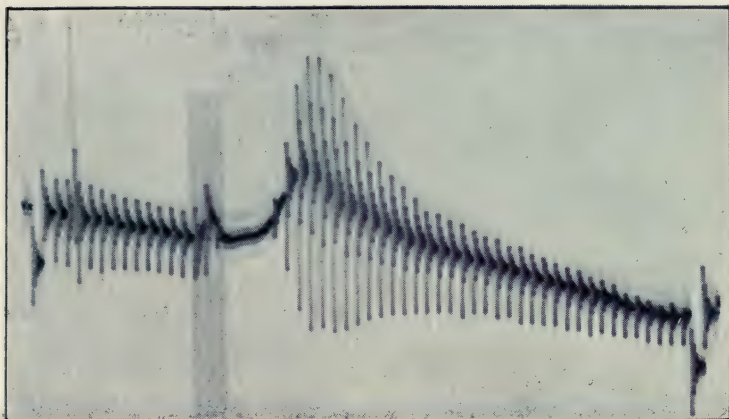


FIG. 4.

Full effect of "much" carbon dioxide on the negative variation. Temporary abolition followed by secondary augmentation.

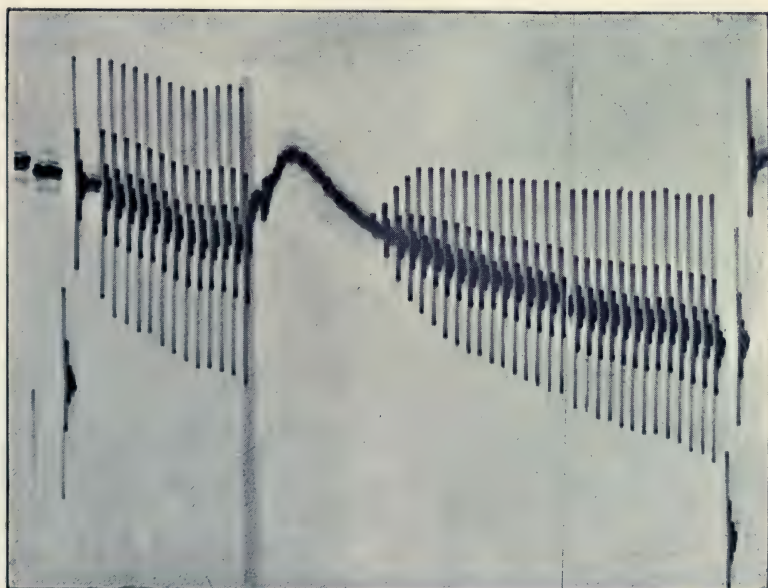


FIG. 5.

Full effect of ether on the negative variation. Temporary abolition followed by recovery.

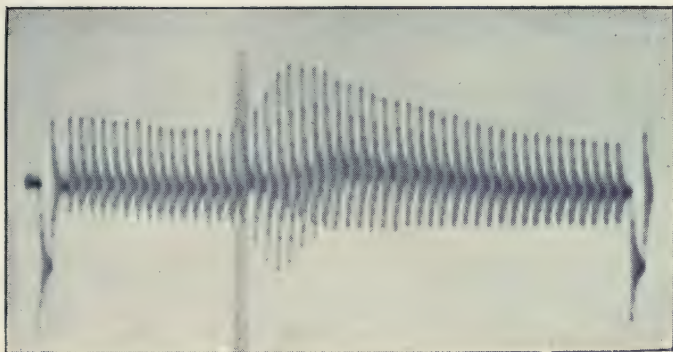


FIG. 6.
Effect of dilute chloroform vapour. Primary augmentation.

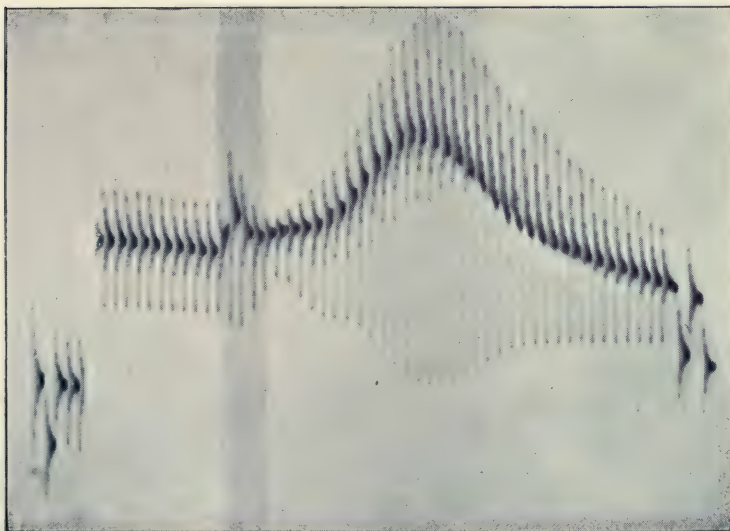


FIG. 7.
Effect of "little" carbon dioxide. Primary augmentation followed by secondary augmentation, separated by a short intervening period during which there is apparently a struggle between augmentation by "little" CO_2 and diminution by "much" CO_2 .

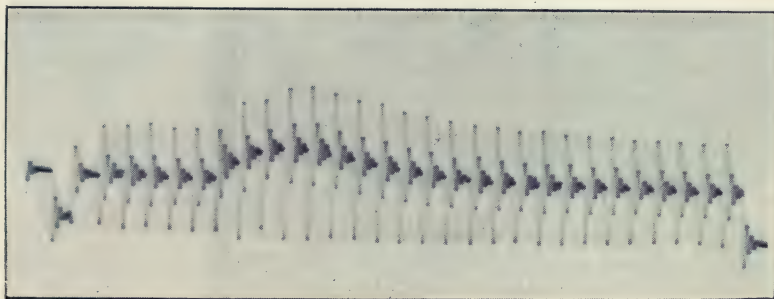


FIG. 8.
Slight effect of ether. Primary augmentation.

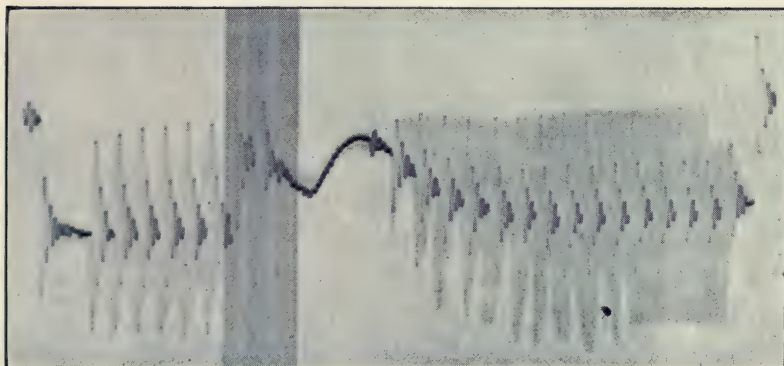


FIG. 9.
Full effect of ether on the same nerve. Primary temporary abolition followed by recovery.

a natural preliminary to any others, for we require to know before testing any gradual effect, what is the gradual effect of temperature alone; what, *e.g.*, may be the difference of effect according as tests are made in summer or winter, at—say 20° or at 5°.

It was indeed an apparently regular difference between my winter and my summer results at these two temperatures respectively, that first made me examine this presumable influence of temperature, for whereas at 5° an almost regular or only slightly diminishing series was the rule, at 20° the series always exhibited more or less decline; nerves were far more enduring during January and February than during July and August, which, indeed, is not matter for surprise.

Experimental raising of temperature (from an initial temperature of 20°) caused a diminution progressing to abolition as the temperature reached 35° to 40°. The nerve was not killed at this last named temperature, for with the subsequent fall to 25° and 20° the variation returned and reached to about three-fourths of its original value.

There are of course many other possible causes of an inconveniently rapid decline—diminution in the strength of the exciting current, which we have tested and found not to diminish under even more severe trial; drying of the nerve, which we have counteracted by the moist chamber; the natural decline of the injury-current, which we have as far as possible escaped by waiting until the current was declining sufficiently slowly;¹ alterations of resistance, which we have

¹ I found later that an excellent means of obtaining a level series of regular effects is afforded by the calcium salts; a one minute bath of a decimolecular solution of $\text{Ca Cl}_2 \cdot 2\text{H}_2\text{O}$ (0.147 per cent.) or preferably in $\text{Sr Cl}_2 \cdot 6\text{H}_2\text{O}$ (0.266 per cent.) secures this often very desirable result. First trials are of course made with a normal nerve, subsequent trials on descending and ascending series induced by a potassium and a calcium bath respectively. A steady and uniform run is of course the most favourable condition of any sort of test, but failing this, it is preferable to test an anæsthetic, or other depressant reagent upon an increasing deflection, a reviving or stimulant reagent upon a diminishing deflection. To the former of these two conditions, strontium chloride was of most welcome assistance, increasing to a steady run the otherwise unworkably declining effects given by the nerves of even the most miserable little frogs. Later still—after I had found that a rapidly declining series was significant of a previous CO_2 effect—I omitted the strontium bath, removed the nerves immediately after decapitation, and placed them until wanted in a decimolecular solution of NaCl .

controlled by graduation ; acidity of the clay electrodes, an accident that has occurred to us, but ought not to occur again ; diffusion of sulphate of zinc through the saline clay to the nerve, which we have practically escaped, by a very long clay plug and by not using the same electrodes for more than one experiment. Finally—and this indeed turned out to be the most essential condition of all—nerves left for any time subject to the reducing action of living tissues, regularly give declining series, this being, as will be shown later, a characteristic effect of CO_2 .

Since the galvanometer is an indicator of *current*, and since current depends upon two factors—electromotive pressure and resistance—it is clear that we must adopt means of determining what alteration, if any, the resistance of our nerve has undergone before we may draw inferences as to alteration of its electromotivity. We shall effect this by a very simple application of Ohm's law, which will afford us incidentally one more justification for a clear understanding of that fundamental physical principle.

Here is an extreme case illustrating the effect of an augmentation of resistance in contributing to a diminishing current of injury and a diminishing excitatory deflection. I give it principally in order to insist upon the necessity of taking a standard deflection before and after each observation.

At outset.				One hour later.			
Current of injury 40	Current of injury	15
Negative deflection by 10 units	10	Negative deflection	3
Standard deflection by 0.001 volt	10	Standard deflection	5

i.e., the resistance was doubled, halving the effect of any given P.D. ; if the P.D. of the currents of injury and of action had not declined, their deflections at the end of the hour with doubled resistance should have been 20 and 5 respectively. As it was they were 15 and 3, *viz.*, a fall of 25 and of 40 per cent. respectively. Except as an illustration of the resistance fallacy the experiment is of no value.

This little resistance box, arranged on the principle of a rheochord, but with a much higher resistance so as to swamp

any alterations of resistance of the cell, delivers to the nerve-galvanometer circuit the pressure of $\frac{1}{1000}$ volt, the deflection of which, recorded at beginning and end of an observation by means of a suitable key, shows whether the resistance of the circuit is high or low, altered or unaltered. If the resistance has diminished during an experiment, the standard deflection will be greater at its end than at its beginning, and *vice versa* if the resistance has increased.

The diminution of the variation by heat is very obviously accompanied by an alteration of the zero value, the line of departure goes north then south. But I place no significance upon this alteration of zero by temperature, for a similar alteration occurs in dummy experiments with a wet thread instead of the nerve, laid upon the leading-off electrodes. I have not experimentally analysed the change into its physical and physiological factors, and have no idea how much mere alterations of resistance contribute to the effect.

I do not, however, mean to imply that in all cases the alteration of zero is to be regarded as insignificant, but that I am not able to perfectly distinguish between an alteration of zero due to a physical modification of the electrodes and an alteration due to a physiological modification of the nerve. That the latter is really in cause is unmistakably proved by a variety of features, among which these are the most noteworthy.

(1) In all the plates, without exception, the inclination of the base line as time went by is from north to south (transverse section of nerve to N. terminal); this decline, steep at the outset and running on concave to the abscissa, has been due to the well-known progressive fall of the current of injury.

(2) On review of the plates exhibiting for a sufficient length of time the effect of a "stimulant" or of a "sedative" drug, it is noticeable that stimulant effects exhibit (in addition to the augmented rhythmic excitatory deflections which have been the principal subject of our attention) an inclination of the base line from south to north, *i.e.*, in the direction of an increased current of injury, while depressant

effects exhibit an inclination from north to south, *i.e.*, in the direction of a diminished current of injury.¹

In prolonged experiments the declining deflection may fall to nothing and then gradually increase in a reversed direction. Without having specially studied this matter I have, in several instances, found this reversal to be attendant upon a reversal of the current of injury. From a theoretical standpoint this should *invariably* be the case; nevertheless, I can remember instances in which the deflection was reversed (*viz.*, north) when the injury current appeared not to be reversed (*viz.*, north of the zero). It is very probable that the decline and reversal of the current of action and the decline and reversal of the current of injury are associated phenomena, dependent upon the act of death gradually progressing in the nerve. Dying tissue is chemically more active than and electrically negative to either normally living or completely dead tissue; at the outset of an experiment, transverse is more dying than, and therefore electrically negative to longitudinal contact, the current of injury is north, and the current of action south; later on when transverse is dead and longitudinal dying, longitudinal is negative, more dying than and therefore electrically negative to transverse, the current of injury is south and the current of action north. But, however this may be, whether this be the correct interpretation or no,² the direction of the currents of action and of injury are immaterial as regards our present purpose. I have not a very large number of observations taken upon a reversed deflection, *viz.*, only ten out of a total of between 400 and 500, for I did not often make tests upon what was to be regarded as a stale nerve, still among these ten I find recorded some of the most characteristic effects—of ether and of chloroform and of strontium namely.

¹ Of the plates here reproduced, fig. 4 best illustrates the augmentation of current, fig. 3, its depression. The two statements made above refer to currents of normal direction.

² Further observations have convinced me that this interpretation is not correct, and that a reversed deflection is independent of a reversed current of injury. Whether it is a true positive variation of the same order as the negative variation of du Bois-Reymond and the positive after-variation of Hering, or an anelectrotonic current aroused by faradisation, must for the present be left open. Whatever its mechanism may be, it is subject to the action of anæsthetics, and therefore presumably a physiological effect.

I should add that my natural prejudice against stale nerve has of late very much diminished. Here is an extreme case of the sort of experience that has had this effect. (Plate, No. 435-6-7.)

FROG KILLED EIGHTEEN HOURS PREVIOUSLY, KEPT IN A MOIST SPACE.

	Deflection.
At outset of observation	— 3·5 mm. (.001 volt = 20 mm.)
Ten minutes after bath of $m/10$ Sr Cl_2	
for one minute	— 3·5 to — 7·5
Ten minutes after second bath	— 23
Twenty minutes after second bath	— 39
Chloroethylene chloride CH_2Cl . $CHCl_2$	
vapour for one minute	0
Thirty minutes later	— 6
CO_2 for one minute	0
Five minutes later	— 9
Thirty minutes later	— 4

It is advisable to use a new gas-chamber and electrodes for each separate experiment with volatile drugs. I mention this point partly as a practical hint, partly in proof of the extreme susceptibility of this method of testing. Using a moist chamber that had been used the day before for a chloroform experiment, on another occasion using one that had been used an hour before for an ammonia experiment, I found that the excitability of the nerve underwent a more or less rapid decline, giving nothing like a normal series such as shown in fig. 1. And in early experiments with solutions I had similar experiences—with potassium salts in particular, also with bromide of gold, and with cocain. So that the comparatively slight recovery that may often occur when a toxic gas, chloroform and hydrocyanic acid in particular, is blown out of the chamber with air is hardly surprising. Another point may be mentioned as being illustrative of the great delicacy of the test. At first I used to clear out the experimental gas sometimes by blowing at the inlet, sometimes by sucking at the outlet, but I soon noticed that the first proceeding was generally more effectual than the second, a result that is intelligible enough when we have witnessed the surprising effect of CO_2 ; by blowing into the gas-chamber, expired air with, say, 4 per cent. CO_2 , was sent in; with sucking, atmospheric air was drawn in. And I

can illustrate this difference to be a reality by an actual example chosen from among many. We may well pause to admire the precision with which the isolated nerve has responded to the chemical difference between atmospheric and expired air, and—what is, perhaps, more immediately to the point—to fit an ordinary india-rubber spray-ball to the inlet of the gas-chamber.

If you wish to promptly and temporarily abolish a large effect, use ether, and use it as freely as you like, the effect will still be temporary. If you wish to instantly and permanently abolish a large effect the best card to play is chloroform. A few puffs of tobacco smoke are probably the readiest means of temporarily augmenting a small variation, or a few bubbles of CO_2 , if the apparatus is at hand, will be even more effective. Always so—in, I may now say, scores of trials *I have never had one failure*, which anyone with recollections of experimental accidents will admit to be a strong statement to make. The augmentation is not however anything like as permanent as the augmentation we shall witness as the result of calcium or strontium; moreover, as indeed you may remember from plate 633 (fig. 4), the augmentation may be secondary to a primary diminution or abolition. It is, in short, a question of quantity, little CO_2 gives primary augmentation, much CO_2 gives primary diminution and secondary augmentation; in any case, augmentation is the salient character of the CO_2 effect.

At first thought, especially in remembrance of Grünhagen's experiment, in which the direct excitability of nerve is abolished by CO_2 , this secondary nerve-reviving effect of CO_2 is rather surprising. Yet our surprise grows less on second thought, especially if we should happen to recollect the drift of Brown-Séquard's admirable but now almost forgotten "rouge et noir" research.¹ CO_2 excites nerve centres; venous blood, or a plus of CO_2 in inspired air causes an immediate increase of respiration, quite apart

¹ Brown-Séquard, "Recherches expérimentales sur les propriétés physiologiques et les usages du sang rouge et du sang noir et de leurs principaux éléments gazeux, l'oxygène et l'acide carbonique," *Journal de la Physiologie*, i., 1858. *Comptes Rendus de l'Académie des Sciences*, xlv., 1857.

from the effect of an oxygen deficit; soda-water is a very common ingredient of restorative drinks; champagne is more exhilarating than hock. You may think that the restorative effect on the human subject of, say, soda-water is in many cases due to the presence of brandy. Admitted, but brandy-and-soda is a more effective stimulant than brandy-and-water.

The next drug to test is obviously alcohol. What will the nerve say to alcohol vapour, or to a mixture of alcohol vapour and CO_2 ? Let us pass alcohol vapour or CO_2 through a wash-bottle with some brandy in it, and see what happens. The nerve approves, if signs of increased excitability be signs of approval; there certainly is an effect, reminding us of exhilaration on the human subject. As in the case of ordinary man, then, the excitability of an isolated nerve is raised by CO_2 by alcohol and by tobacco smoke.

Pray do not imagine that I am offering any plea for (or against) the use of alcohol and tobacco; my business is merely to observe and compare phenomena *inter se*. Besides, here again the question of quantity in relation to excitability comes in; I might put side by side plates of alcohol and of calcium phosphate to plead for alcohol, and plates of alcohol and hydrocyanic acid to plead against alcohol. Here they are, but notice the alcohol strengths—10 per cent. in the “evidence” for alcohol, absolute in “evidence” against alcohol.

Do not let me, however, be taken as urging to extremity a comparison between the properties of an isolated nerve and those of a human organism. Obviously a nicely-adjusted organism, with organs of many grades of susceptibility, including nerve centres of the highest susceptibility, is not adequately represented by a single tissue, such as nerve, which, as we have seen, tolerates interruptions of blood supply and holds out against wear and tear to almost incredible extremities. Clearly the excitabilities of a man's nerve centres may not be taken as represented in miniature by the excitability of a frog's nerve. We may study on parallel lines the comparative efficacy of such drugs as

alcohol, chloroform, ether on the living organism and upon the living tissue, and from their scale of action on the latter draw conclusions as to their scale of action on the former, but we may not pass from one line to the other and conclude that because nerve tolerates a given poison, a man's nervous system will equally tolerate that poison; we may not, for instance, argue that because a nerve will go on without blood and without oxygen, a man will be similarly independent. All we may do is to conclude from the action on nerve to a similar action on the nervous system, and to argue from the differential action of two drugs upon living nerve to a similar differential action on living man; the scale of quantity and time may be, and most probably is, very different from that on which our nerve data have been observed. Take CO_2 , for example, we find that in small quantity it raises excitability, that in large quantity it depresses, then raises excitability in the case of nerve; we may fairly infer that it will have similar effects upon the nervous system of a man, effects that will declare themselves in the parts of greatest susceptibility; but we may not conclude that a man will tolerate CO_2 in quantity or for periods that are found to hold in the case of nerve. Take N_2O again; we find that, as compared with CO_2 , it has no action on nerve, we may conclude that on man it has, at any rate, less action on the nervous system than has CO_2 . Take ether and chloroform; we find on nerve that the latter is far more toxic than the former, we may infer that a similar difference will obtain on man. And having traced a parallelism in cases well known such as these, we may proceed further, and, in the case of a drug of unknown or of doubtful action, use a nerve test as at least a preliminary question—and—answer in exact terms pointing the way. Find an antagonist to chloroform on a frog's nerve, and you will in all probability have found an antagonist to chloroform on a man's nervous system. Do not either mistake the nature of the argument, as, for instance, by taking the action of chloroform on excited nerve as an evidence that chloroform anæsthesia is due to an action on a man's intact nerves; chloroform acts upon all living matter—upon vegetable as

well as upon animal protoplasm; when a man is chloroformed, chloroform acts upon all his tissues, but takes effect by virtue of its action upon his most susceptible parts—upon his highest nerve centres first, then his lower centres, may-be his medulla, may-be his heart, and in all likelihood he is perfectly inexcitable as to his brain long before his nerves have been sensibly affected. Anything having power over nerve will have power over nerve centre, but the converse does not hold good; an alteration sufficient to excite or to paralyse a nerve centre is not necessarily sufficient to excite or paralyse a nerve.

And there are many considerations to be borne in mind in connection with any method by which drugs are brought into immediate relation with isolated tissues and organs; not as many, indeed, as under the complex conditions presented by a human organism into which a drug or a collection of drugs is experimentally poured, but still many, and among them considerations that bid us pause before transferring results of such experiment forthwith to therapeutics.

Above all, the drug is applied in a big dose and rapidly to a tissue as compared with the gradual diffusion of a much smaller dose diluted in the mass of blood, chemically altered, it may be, by the digestive fluids or by the living organs. We classify drugs as stimulant and sedative. Can you name many "sedative" drugs that do not on occasion stimulate, or one "stimulant" drug that, if pushed, does not exhaust and depress? Consider some of our best known drugs—morphia, alcohol, chloroform, ether, strychnia. Morphia a sedative, alcohol a stimulant, chloroform and ether anæsthetics, strychnia a convulsant. These are the prominent effects of the drugs respectively under their ordinary modes and conditions of exhibition to the human subject, but our knowledge of their therapeutic value could never have been a mere translation to the human subject of facts ascertained on frogs or dogs or rabbits.

I may seem to be expressing distrust of laboratory facts; that is, however, not my meaning, which is that such facts must be intelligently digested and intellectually integrated,

not merely swallowed whole in the laboratory to be served up whole at the bedside.

What we can learn as regards the action of a drug upon an isolated living, that is to say excitable, tissue is in principle whether its predominant action is to excite or to suppress activity. A drug that has any action at all on the tissue must produce one or other of these effects, and, *a priori*, might be expected to produce both effects. Most sedative drugs in the earlier stages and at lower doses are stimulant; all stimulant drugs in the later stages and at larger doses are depressant. Opium is a stimulant before it is an opiate, brandy is an opiate after it has been a stimulant.

By the direct application to nerve of any drug having action upon nerve, we may expect to witness a depressant action; and it will only be in exceptional cases or in cases where the drug is applied cautiously and gradually that we shall also witness a stimulant action. Even bromide of potassium, the predominant effect of which is depression, may be shown to produce a preliminary stimulation.

The practical rule as regards procedure towards isolated nerve seems to me to be as follows—taking for granted that the obvious precaution has been observed of applying the as far as possible *isolated* drug—namely, to observe the first smallest producible effect as well as the ultimate effect of comparatively large doses.

The latter was all we did above, and we found that the main effect in each case was depression of function, but did we go too fast with the intoxication, did we miss a preliminary stage of excitation? That can only be determined by carefully examining for the first smallest producible effect.

What we may expect to find is that any “stimulant” drug if pushed, becomes depressant, but we may also admit as reasonably probable that drugs should be encountered with an *ab initio* depressant action.

We have a familiar instance of a stimulant followed by a depressant action in the case of alcohol. Here is another instance which has occurred in one of my preliminary trials;

testing the nerve by strong ammonia, I obtained an immediate loss of excitability; testing with weak ammonia—in this instance a chloroformed nerve to see whether ammonia had any restorative action upon such a nerve—I obtained an augmentation of excitability; in fact, whereas the chloroformed nerve gave no effect, it did give a distinct effect when weak ammonia vapour was blown through the gas-chamber.

This matter of chloroform and ammonia has interests of a practical character, and I therefore repeated it more than once, varying the strength of chloroform and of ammonia, and submitting the nerve to chloroform after ammonia, or to ammonia after chloroform—or to both vapours simultaneously—with the view of bringing out the probable antagonism between them.

But more experiments are required before I may venture to make positive statements of detail on this point. All that I may legitimately do at present is to lay before you items of evidence in illustration of the stimulant and depressant action of ammonia, of chlorine, of chloroform and of ether.

Here is a plate (No. 191) exhibiting the action of “strong” ammonia vapour upon a normal nerve; the series of deflections before ammonia are quite regular, and on the plate have an amplitude of 25 mm., after ammonia they are still regular, but with an amplitude of less than 1 mm.

Here is another plate (No. 209) exhibiting the action of “weak” ammonia vapour upon a nerve completely paralysed by the previous administration of chloroform vapour.

PLATE 209.

Normal deflection = 8 mm. (regular).
 Deflection after CO_2 = 30 mm. (regular).
 Deflection after CHCl_3 = 0 mm.
 Deflection after NH_3 = 5 mm. (declining).
 10 minutes later = 0 mm.

As you may gather by comparing this plate with other chloroform plates, the recovery is obviously not spontaneous, but provoked by ammonia; moreover, as you see, it is not complete nor permanent. Ten minutes later the deflection has fallen again to zero. Perhaps there was too

much chloroform, perhaps there was too little ammonia. However that may have been, here is another observation in which there was certainly less chloroform administered, and possibly more ammonia, in which the ammonia effect is perfectly clear (242). And here is a similar observation with regard to ether (243). With chlorine water (B.P.) I had precisely similar results; diluted twenty times it was stimulant, undiluted it was promptly depressant.

A more important point, however, with regard to chloroform is the antagonistic effect of CO_2 . Run CO_2 alone through the nerve-chamber for one minute, and after a minute or two of depressed excitability you will witness the usual augmentation. Run chloroform vapour alone through the nerve-chamber, for one minute, and you will witness the usual abolition of excitability, and very probably the abolition may be final. But run the two together by joining up the CO_2 apparatus with the CHCl_3 bottle, and you will very probably not have to wait long before witnessing a more or less well-marked recovery.

This idea of the stimulant and depressant action of a drug will best be brought clearly to mind by comparing the behaviour with regard to nerve of a group of drugs the action of which has been most abundantly observed upon the human organism, viz., alcohol, chloroform and ether, which together form the now well-known "A.C.E." mixture of anæsthetists. In this case we know already that each of these three reagents are stimulant and depressant, that of A. the salient effect is stimulation, of C. depression, and that in the case of E. the stimulant effect is more marked than in that of C., less marked than in that of A., while the depressant effect of E. is more marked than in that of A., less marked than in that of C.

The evidence of isolated nerve affords precise and elegant confirmation of this result of prolonged experience, and being thus in harmony with such experience signifies that we are in possession of a truthful method. In a series of experiments with different strengths of alcohol, chloroform and ether, respectively, the results come out in a most clear and cogent form, exhibiting as in a nutshell the comparative stimulant and depressant actions of A., of C. and of E.

Here are *e.g.*, the essential data to the point, obtained in a group of five consecutive experiments *ad hoc* upon as many nerves in gas chambers as represented in fig. 2, the reagent being in each case blown in *per* wash-bottle during a period of one minute.

FIVE EXPERIMENTS RE STIMULANT AND DEPRESSANT ACTION OF ALCOHOL, CHLOROFORM AND ETHER.

Time.		Deflection.
1.	0— 5 mins. 6—30 „	Undiluted chloroform 25 mm. (regular). Abolition without preliminary aug- mentation. 0.
2.	0— 5 mins. 6—15 „ 15—30 „	Undiluted ether 14—13 mm. Abolition without preliminary aug- mentation. Trace to 4 mm.
3.	0— 5 mins. 5—10 „ 10—20 „ 30—40 „	Undiluted alcohol 20 mm. (regular). 20—25 } <i>i.e.</i> , augmentation followed 21—19 } by diminution. 16—14 }
4.	0— 5 mins. 5—10—15—20— 25—30 mins.	Chloroform 1 per cent. 11 mm. (slightly augmenting). 11—27—22— } <i>i.e.</i> , augmentation fol- 15—8·5—5—3 { lowed by diminution.
5.	0—5 mins. 5—10—15—20— 25—30 mins.	Ether 1 per cent. 12 to 8 mm. 8—17—14—11 } <i>i.e.</i> , augmentation fol- —9—7—5—3 } lowed by diminution, both less pronounced than in exp. 4.

And we are justified by such experiments in giving form to our knowledge of the three drugs in this graphic summary, which might also be taken to represent what is admitted to be their action upon the human subject (fig. 10).

Besides these familiar anæsthetics I have (as a preliminary to more systematic examination, and as a further test of the applicability of the method) tried the effects of some of the simpler and less commonly used anæsthetics, such as the chloride, bromide and iodide of ethyl, and several of the various chloromethanes, and chloroethanes that have been tried as anæsthetics.

Here is a memorandum of the anæsthetics whose actions I have compared *inter se* by the method of isolated nerve, with a memorandum of what is probably one of their physical properties by which their physiological effect is most influenced, viz., their boiling-points.¹ I have to

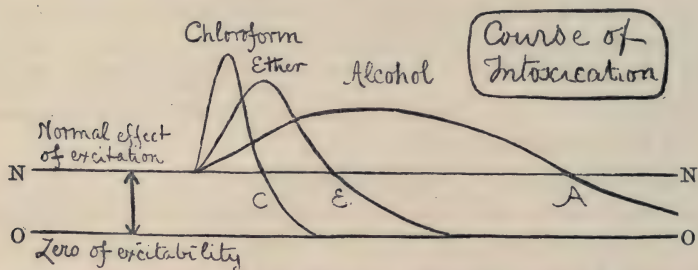


FIG. 10.—Diagram of the excitatory and depressant effects of chloroform, ether and alcohol.

O—O represents the level of zero excitability.

N—N represents the level of normal maximal excitation, and the three curves the stimulant—depressant effects of chloroform, ether and alcohol respectively.

say, however, that I have as yet made no definite tests of this influence, but have only seen in a general way that temperature influences the effect, as indeed do many other circumstances, such as the quality of the nerve, and the reagents to which it may have been previously submitted.

CH_3Cl	..	Chloromethane	or Methyl chloride	..	-23.7° .
CH_2Cl_2	..	Dichloromethane	„ Methylene chloride	..	41.8° .
CHCl_3	..	Trichloromethane	„ Chloroform	..	61° .
CCl_4	..	Tetrachloromethane	„ Carbon tetrachloride	..	76.74° .
$\text{C}_2\text{H}_5\text{Cl}$..	Chloroethane	„ Ethyl chloride	..	12.5° .
$\text{C}_2\text{H}_5\text{Br}$..	Bromoethane	„ Ethyl bromide	..	38.4° .

¹ CHCl_2 , CH_2Cl_2 should have been omitted from this list as I have not yet obtained it.

C_2H_5I ..	Iodoethane ..	or Ethyl iodide	72°.
CH_2ClCH_2Cl	Dichloroethane	„ Ethylene chloride or Dutch liquid ..	83·5°.
CH_3CHCl_2 ..	Dichloroethane	„ Ethyldene chloride ..	60·1°.
$CH_2ClCHCl_2$	Trichloroethane	„ Chloroethylene chloride	114°.
CH_3CCl_3 ..	Trichloroethane	„ Methyl chloroform ..	76°.
CH_2ClCCl_3 ..	Tetrachloroethane	135°.
$CHCl_2CHCl_2$	Tetrachloroethane	„ Acetylene tetrachloride	147°.
$CHCl_2CCl_3$..	Pentachloroethane	158·9°.
$(C_2H_5)_2O$..	Diethyl oxide	„ Ether	34·6°.
C_2H_5HO	„ Alcohol	78·5°.

It is obviously impossible for us to pass all the records of these sixteen reagents through the lantern, nor would such a running review be a very suitable means of forming a conclusion as to their relative efficacy as anæsthetics.

But from the records laid out in order on that side-table, you may, after leisurely inspection, decide for yourselves whether or no the conclusions I am about to draw from them are justified.

First let me ask you to make a distinction between the activity of these reagents with respect to toxicity and their activity in reference to the chief desideratum of an anæsthetic, viz., degree and rate of recovery from profound but not definitive inexcitability. A drug may quickly abolish excitability, but it is toxic rather than anæsthetic if after its removal excitability does not return.

In this respect you will at once pick out the trichloromethane, $CHCl_3$, more familiar to you, perhaps, under the name of chloroform, as of all these drugs in regard to the excitability of isolated nerve, the most *toxic*. You will as unhesitatingly pick out as the apparently most promising *anæsthetic*, the ethyl dioxide $(C_2H_5)_2O$, *i.e.*, ordinary ether or “sulphuric ether.” The crowd of records would then to your eye dispose itself in two groups, into the chlorinated group with chloroform at its head, and toxicity as its characteristic action, and the non-chlorinated group with ether at its head, and anæsthesia as its characteristic action. There are indeed two exceptions to this law, the methyl chloride CH_3Cl , and the ethyl chloride C_2H_5Cl , which, as you see, fall into the ether group, but the exception is

only apparent, for looking to the two ethereal salts C_2H_5Br and C_2H_5I , you at once conclude from the similarity between the three that C_2H_5Cl is anæsthetic by virtue of the C_2H_5 radicle rather than by the Cl , Br or I appendix—the radicle C_2H_5 is the predominant partner. C_2H_5Br and C_2H_5I I have not yet tested, but feel very confident that they would conform to and confirm the law.

Further than this I hardly care to go, although there is much temptation to make further and premature comparison between individual members of the methane and ethane groups and the ethereal halides. But to impress what surely must arouse most serious reflections on a matter very near and practically important from the clinical side, I will pass through the lantern one pair of plates representative of fifteen other similar pairs registered upon fifteen nerves submitted for one minute to the action of ether and of chloroform respectively.

Comment is needless. One is rather inclined to diminish the stress that such documents must produce on the mind by saying: but this is only a frog's nerve, its current of action is a recondite phenomenon, not necessarily co-extensive with physiological action; a nerve fibre is not a nerve cell. But all possible discount made, the fact remains that here in the nutshell of a laboratory experiment lies sharp and concentrated evidence similar in character to the more diffuse, and therefore less pointed evidence of clinical experience.

You will say perhaps, "but your nutshell evidence proves too much; you show that chloroform easily kills nerve, but we know that chloroform only now and then kills a man or woman." Granted—granted also that a naked nerve is a delicate test, that it returns in exaggerated terms its verdict in the ether *v.* chloroform issue. But every anæsthetist—I will not say "chloroformist"—will tell you that a chloroformed patient is hovering between life and death, and that the utmost watchfulness is needed to keep him on this side of a boundary which he sometimes crosses in spite of—or it may sometimes happen—by reason of that watchfulness. Well, the analogy between the clinical event



Fig. 11.

Effect of ether, then of chloroform. The latter administered with great care in quantity sufficient to cause temporary and not permanent abolition of excitability.

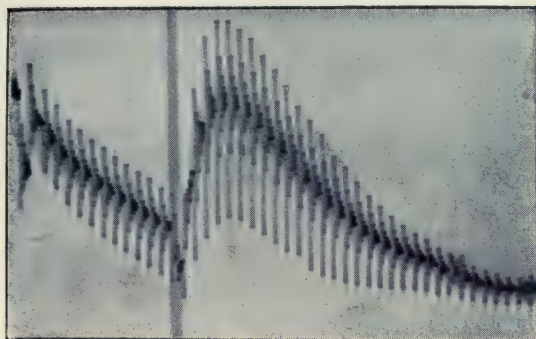


FIG. 12.

Effect of chloroform administered too dilute for anæsthesia, and producing primary excitation passing gradually into a permanent abolition of excitability (from which the subsequent administration of weak ammonia produced only a slight and temporary recovery).

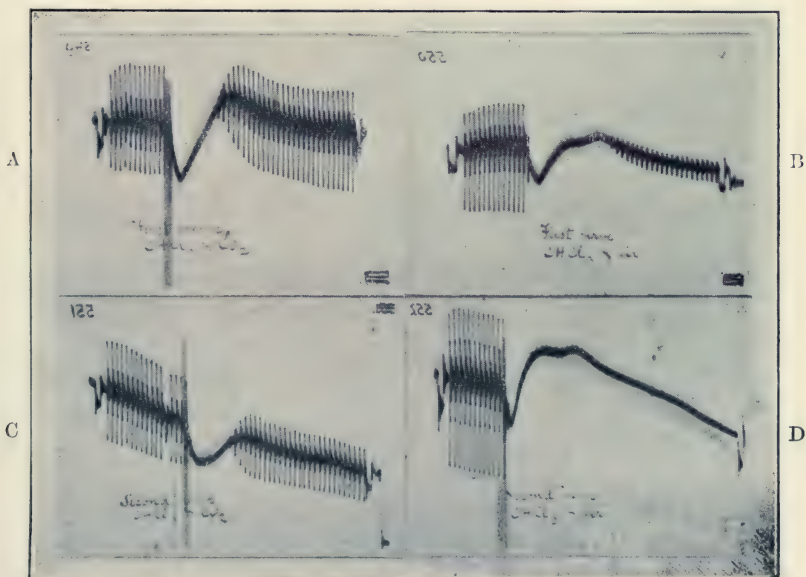


FIG. 13.

- A. First nerve, chloroform with CO_2 .
- B. First nerve, chloroform alone.
- C. Second nerve, chloroform with CO_2 .
- D. Second nerve, chloroform alone.

and the laboratory experiments holds even here, only the naked nerve being a finer touchstone than an entire organism irrigated by blood vessels and ventilated by lungs, it is more difficult to *temporarily* anæsthetise by chloroform a nerve than a man. Still it is possible, and I now show you the best instance of successful anæsthesia of nerve I have obtained by carefully graduated chloroformisation (fig. 11). The experiment is a difficult one, and its difficulty illustrates, I think, two opposite dangers of clinical practice—the Scylla “too much” and the Charybdis “too little.” You may give your nerve too much, and thus kill it; you may give your nerve too little, and it is, without ever having manifested any sharp suppression of anæsthesia, slowly and steadily drugged to death. Here is such a case: the nerve has been submitted to dilute chloroform vapour, and after manifesting the augmented excitability characteristic of the weak drug, it has steadily died without exhibiting any true anæsthetic state (fig. 12). Thus a nerve—or it may be a patient—can be suddenly struck down and killed by too concentrated chloroform, slowly pressed down and killed by too weak chloroform; just that blow that shall stun without killing is the most difficult to deliver—on nerve at any rate. As far as my experience goes the greater danger—on nerve again—is the “too much” rather than the “too little.”

The case of ether has been quite otherwise, using the same delicate test-object of living nerve. I have found it comparatively easy to *temporarily* suppress excitability and to maintain it suppressed during long periods, and it has happened in only one or two instances of “poor” or actually moribund nerves that the suppression has not been succeeded by complete recovery.

As the case stands clinically with respect to ether *versus* chloroform, the question is presented as a *comparison* of two drugs, one more dangerous and more convenient than the other, and an attempt to determine from what quarter danger is most to be expected in the case of the former. That is how the case stands in England; we tabulate death rate statistics, and we appoint commissions

to debate whether chloroform kills by the heart or by the lungs. In American clinics, and in the direct experiments now before you, the comparison is a *contrast* between a safe and an unsafe drug, an anæsthetic and a poison.

It is of course quite beyond my province to discuss in detail the reasons that might on occasion justify the preference of the unsafe drug, more especially as regards the relative gravity of after effects, nor am I disposed to discuss the "safeguards" under which the unsafe drug may be employed for every day purposes, inclusive of minor surgery.

Still, having regard to the fact that on occasion it may be necessary and therefore legitimate to employ the unsafe, but more powerful drug, I will mention two points of detail as the outcome of my nutshell experience.

(1) The idea that chloroform is dangerous according to its impurity, innocuous according to its purity, is erroneous. Pure chloroform free of ethereal impurities is more toxic than impure chloroform containing such. The well-known A.C.E. mixture is less toxic than chloroform alone, more toxic than ether alone.

(2) With regard to the opposed systems of administering chloroform: (a) with much diluting air; (b) in stronger vapour and exclusive of air; experiments on isolated nerve, *pro tanto*, indicate the second as the less dangerous mode of procedure. CO_2 in the nerve-chamber (and *possibly* therefore an excess of CO_2 in the lungs and in the blood acting upon the nerve centres) is in its effect an adjuvant of the anæsthetic action and an antagonist of the toxic action of CHCl_3 vapour, although it is very evident that obstruction to free aeration, at a time when the blood is charged with chloroform and the organism verging on the limit of "too much," must add to danger, whatever advantage might *theoretically* be hoped for from the primary and secondary effects of CO_2 brought into action at the outset of anæsthesia (fig. 13).

Carbonisation during the production of anæsthesia, free aeration during the return from anæsthesia are, as regards isolated nerve, conditions *pro* the anæsthetic, *contra* the toxic effect of CHCl_3 .

You have, no doubt, learned to carefully remove all obstruction to free aeration during anæsthesia; I cannot venture to advise you to abandon such care; think the matter over, test the point on animals perhaps, before you finally make up your mind how—if chloroform must be used—it may be given with least imprudence.

You may, perhaps, be inclined to say that I have already transgressed the limits of strict experiment, and trespassed by inference upon the domain of clinics. That may be, but I shall defend myself by urging that having once reported experimental results of such obvious practical bearings, I am bound to give the inference as it presents itself to my mind, rather than leave it to be drawn less guardedly and cautiously than seems to me to be desirable. Let me then clearly and explicitly state that I regard these experiments (1) as in high degree confirmatory of the almost universally admitted greater clinical danger of chloroform, and (2) as indicative of a possible good effect of CO_2 in the production of chloroform anæsthesia. And as regards this second inference of possibility, let me expressly add that I do not recommend its trial upon the human subject; at this stage it is only indicated as requiring careful trial upon cats and dogs.

ACTION OF REAGENTS ON ISOLATED NERVE.

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(To be continued.)

FURTHER OBSERVATIONS ON THE EXCRETION OF URIC ACID IN EPILEPSY AND THE EFFECTS OF DIET AND DRUGS ON THE FITS.

BY ALEXANDER HAIG, M.A. AND M.D.OXON, F.R.C.P.,

Physician to the Metropolitan Hospital and the Royal Hospital for Children and Women.

EVER since I published in 1888¹ my first observation on the excretion of uric acid in epilepsy I have been asking myself this question: "Is the uric acid fluctuation a cause or a consequence of the epileptic fit?" And it seems to me that from whatever point of view I look at it, whether I regard the relation of the excretion to the fit, and the fact that the plus excretion of uric acid is the result of a minus excretion which preceded the fit, and could not, therefore, possibly be caused by it: or whether I regard the extensive and important series of parallels between an epileptic fit and a uric acid headache, or when I look at the very powerful effects of uric acid on the circulation, nutrition, and function of many important organs and tissues, I seem to get only one answer from all sides, "Cause."

Now it follows that if uric acid is the cause of the fits we ought to be able by controlling uric acid to control the fits to something like the same extent to which, by controlling the uric acid, we can control the uric acid headache (migraine).

Now my own headaches, and those of quite a large number of fellow-sufferers have been almost absolutely controlled by diet, which controls the uric acid; and the more completely the uric acid is kept out of the blood the more

¹ *Neurologisches Centralblatt*, March, 1888.

completely does the headache remain absent; but the moment that uric acid is introduced into the blood in any quantity the headache begins once more.

Thus my own headaches have fallen from forty to fifty in twelve months down to four or five in the same period, and even these four or five are the results of quite definite introductions of uric acid which could be prevented if greater and more constant care in diet was possible.

I may claim, I think, then, for this headache (migraine) that I have absolutely and completely controlled the attacks both in myself and many others, by clearing the blood of uric acid and keeping it clear.

Can the same be done for epilepsy? My impression is that it will certainly be more difficult to do, but should not be by any means impossible, though the absolute amount of control may be less in the case of the fits than in the case of the headaches.

In headaches there is a large and comparatively slow fluctuation in the excretion of uric acid, *i.e.*, a man has a headache to-day because yesterday in the alkaline tide something interfered with his urate excretion, and he passed out of his body only one half the quantity he usually passes in the same hours; to-day the uric acid held back yesterday adds itself on to the natural excretion, and there is consequently enough in his blood for a few hours to affect blood pressure and the intracranial circulation.

The object I attain by diet is so to reduce the intake and formation of uric acid that the largest quantity he is likely to get in his blood as the result of such a fluctuation, shall never, or only very rarely, be sufficient to affect the blood pressure and the intracranial circulation to a serious extent.

Now in epilepsy (as will appear from my clinical results further on) the fluctuation is shorter and steeper; for two to three hours there is a very powerful retention of uric acid, during which, just as in the corresponding case of headache, the patient feels unusually well and strong; and this is followed in the next two hours by an equally short and sharp plus excretion of all the uric acid held back.

In a word the epileptic wave of excretion runs very high,

but only for a short time, and it is very difficult to keep the general level of uric acid excretion so low that these waves shall not occasionally get high enough to do damage by raising the blood pressure and affecting the intracranial circulation.

I note with much interest in this connection that Dr. Gowers remarks (*Lancet*, 1895, vol. i., p. 1626) that the visual sensations of epilepsy last seconds, those of migraine minutes, while the convulsion of epilepsy lasts minutes, and the paroxysm of migraine hours.

With regard to the parallels between the uric acid headache and the fits of epilepsy, though much has been written on the subject it may not be altogether without interest if I shortly mention the main points here.

Now these two diseases agree in—

- (1) Having no morbid anatomy (1).
- (2) Being periodical.
- (3) Occurring for years or for life.
- (4) Occurring together or in alternation.
- (5) Being preceded by a minus excretion of uric acid accompanied as usual by feelings of well-being.
- (6) Being accompanied by a plus excretion of uric acid, and bearing a generally similar relation to the natural diurnal variations in the excretion of uric acid.
- (7) In being worse at or about the menstrual period in women.
- (8) In being better during pregnancy and worse after delivery (2).
- (9) In being better during fevers and worse after them (3).

In (7), (8), and (9) they both exhibit a remarkable parallel with another disease which is due to uric acid, viz., Raynaud's disease (see "Uric Acid," third edition, p. 353).

(10) In being preceded by high blood pressure (4) and irregular pulse, with coldness and numbness of the extremities and shivering.

(11) In bearing similar relations to gout, rheumatism, and other uric acid diseases.

(12) In being preceded or accompanied by subnormal temperature.

(13) In being followed by a fall of blood pressure and a diuresis (5).

(14) In being similarly related to dyspepsia and gastro-intestinal disturbance however caused.

(15) In being similarly affected by weather and changes of temperature.

(16) In being accompanied by more or less complete suspension of gastro-intestinal digestion and absorption.

(17) In being accompanied or followed by albuminuria (6).

(18) In being preceded by marked changes in or even complete loss of sight (7).

(19) In being similarly related to peripheral neuritis (8).

(20) In being made worse by cold.

(21) In being made worse by administration of quinine, iron (9), or lead.

(22) In being improved by nitrites, acids, or antipyrin (10).

(23) In being relieved by compression of the carotid arteries.

(24) In being more or less completely cured by diet.¹

With regard to (6) it is remarkable how these two diseases, in common with mental depression, angina, and other similar troubles,² tend to attack sufferers in the early morning hours; just as the acid tide of the night is coming to an end, just as the uric acid, which has been for many hours below urea tends with the falling acidity to rise above it, just as the low blood pressure, relaxed arterioles, and quick pulse of the night give place to the high blood pressure,

¹ I give a few references with the above statements in cases where I have not previously given them; but most of the points will be found to be discussed at length in my book, to which I must refer for further information.

(1) Wynne, *Lancet*, 1893, vol. ii., p. 434. (2) Oliver, *Lancet*, 1894, vol. i., p. 1295. See also *British Medical Journal*, 1891, vol. ii., pp. 1120 and 1236. (3) *Lancet*, 1894, vol. ii., p. 1438. (4) Féré, *Progrès Medical*, 1889, vol. i., pp. 415 and 434; and vol. ii., p. 26. (5) Féré, *Progrès Medical*, 1888, vol. ii., p. 452. (6) *Archiv de Neurol.*, May, 1892. (7) Gowers, *Lancet*, 1895, vol. i., p. 1626. (8) Charcot, *Progrès Medical*, 1890, vol. ii., p. 83; and Cagney, *Lancet*, 1891, vol. ii., p. 1095. (9) Radcliffe, *Practitioner*, 1883, vol. xxx., p. 95. (10) *Lancet*, 1887, vol. ii., p. 1163.

² "Uric Acid," third edition, p. 441, where I quote the interesting observation of Prof. Mays that angina, just like migraine and epilepsy, may be preceded by feelings of well being, which is the proof of a uric acid fluctuation.

contracted arterioles, and slow pulse of the early morning; and these facts have been observed by hundreds of people who did not know that the uric acid fluctuation was the cause of all the other changes, and thus missed the fact that explains them all, and proves that uric acid is the cause of all these diseases.

Here, then, in the early morning hours we have a fluctuation in the excretion of uric acid very similar to that which occurs in the epileptic fit, and it is little wonder that a slight pathological increase of this fluctuation should suffice to produce any or all of the above diseases.

In the third edition of my book I have figures to show all these natural fluctuations in the daily excretion of uric acid, and also those which accompany menstruation in women, and explain the relationship between that function and those diseases; and their relation to pregnancy and fevers admits of similar and simple explanation, but it would lead us too far from our subject to discuss these matters fully here.

With regard to (14) the relation between migraine and dyspepsia is that the dyspepsia precedes the headache and is its cause for dyspepsia, causes a fall of urea, and of the acidity of the urine, and the fall of acidity means that the alkalinity of the blood is increased, and it is immediately flooded by any uric acid available for solution; but if you control the uric acid the dyspepsia will have little or no effect, and further than this it appears, for reasons which will be found in "Uric Acid," third edition, chapter x., that the dyspepsia is itself, to a large extent, due to the presence of excess of uric acid in the body and blood, so that when you have removed uric acid you will have removed both the dyspepsia and the headache which it precipitates. I believe that the connection between these phenomena in relation to the fit of epilepsy is precisely the same as in the case of the uric acid headache.

In all these respects there is the very closest parallel between the uric acid headache and the fits of epilepsy or uræmic, puerperal, and other convulsions.

If both are due to uric acid nothing is more simple than

the causation of this extensive parallelism, but if not it is absolutely inexplicable.

There has recently appeared in the *Archives de Neurologie*,¹ an extremely interesting series of papers by Drs. J. Voisin and R. Petit on Epilepsy as due to Intoxication.

They do not appear to have seen my previous writings and researches, nor have they, as yet, identified or named any poison, so that in this respect I am considerably ahead of them: but their argument for intoxication strongly supports my own, and, moreover, I can show that almost every point on which they lay stress can be absolutely and completely explained if it be granted that uric acid is the toxic substance; and those who have watched my results in these and other directions, will, I hope, have no difficulty in admitting that it is toxic.

I shall now only mention one or two points in their papers that bear on uric acid as the cause of epilepsy.

Thus they lay great stress on the occurrence of gastric troubles before and during the fits, or series of fits: but if the series is coming to an end then the tongue clears and the gastric symptoms subside, but if, on the other hand, the gastric symptoms do not subside, the epileptic phenomena will continue.

The explanation of this is simple enough when we know that every gastric upset brings excess of uric acid into the blood in those who have much of it in their bodies.

The gastric trouble is thus the cause of the uricacidæmia which is the cause of the fits, the malaise, the headache, and all other symptoms, and it (uricacidæmia) in its turn intensifies the gastric upset, and adds putrefaction with possible formation of toxic products.

But the gastric troubles are the result of intoxication by uric acid, even though their result is to increase the toxæmia, for in migraine if you keep the blood clear of uric acid both the gastric troubles and the paroxysm of headache are absent.

With regard to the urine, they point out that there is diminished toxicity before and during and increased toxicity

¹ *Archives de Neurologie*, 1895, Nos. 98, 99, 100, et seq.

after the fit, and it is interesting to note the exact parallel between the excretion of uric acid (which the authors have not estimated, though they notice in one case a deposit after a fit) and the toxic substance.

The authors also mention an extremely interesting case, in which a wound that was being dressed stopped bleeding with the onset of a fit, and no pulse could be felt at the wrist: but at the end of the spasmodic stage the pulse reappeared, beating 142, the blood pressure rose, and the wound began to bleed freely once more.

In this case the fit was probably due to stoppage of the heart, which failed before the high blood pressure; and with the fall of blood pressure due to the stoppage of the heart, the circulation practically ceased and the wound stopped bleeding, and the corresponding stasis in the cerebral circulation produced the convulsion; later on, the pressure having fallen, the heart recovered itself and began beating again (see "Uric Acid," third edition, pp. 168 and 210).

Then, after the fit, the pressure rose above normal, though probably not so high as it was just before it, when the heart failed.

I have seen a very large number of cases in which the heart has given obvious signs of impending failure before the high blood pressure produced by uric acid in the uric acid headache (migraine); and these cases differ from the above case of epilepsy merely in the fact that the heart, though threatening to fail, did not fail, and so there was no convulsion. And such severe paroxysms of headache may, just like an epileptic fit, be followed by albuminuria.

Talking of albuminuria reminds me of paroxysmal albuminuria and hæmoglobinuria, which I have long held that this albuminuria resembles ("Uric Acid," third edition, pp. 388 and 391); and so of the blood changes which take place in paroxysmal hæmoglobinuria and anæmia as the result of the presence of an excess of uric acid in the blood stream; and I have therefore been very greatly interested to see that the authors mention that a diminution of hæmoglobin and an increase of hæmatoblasts has been observed in connection with the fits of epilepsy, for this is a change

which I thought might very probably occur in the blood at the time of a fit, because as I have pointed out ("Uric Acid," third edition, p. 366), my own blood varies its quality from day to day, in accordance with the amount of uric acid passing through it.

I had thought of this in the case to be presently related, and Dr. Huntley very kindly examined her blood after one or two fits; but his results were indefinite, and I was unfortunately not able to examine it myself; but I hope to do so in some future cases, as I have yet to learn at what length of time after the fit the blood changes show best: probably after or during the albuminuria, when present.

The authors also point out that the repair of wounds is defective while the epileptic phenomena continue, and only goes on properly after the toxine has been eliminated.

Here, again, we have the work of uric acid, for I have for years been pointing out that opium, which the surgeons use to aid the repair of skin wounds, acts by clearing the blood of uric acid, and thus allowing a free capillary circulation in the skin, which is impossible so long as the blood is full of uric acid; and in the ordinary uric acid headache the signs of defective capillary circulation throughout the body are so obvious that they can hardly be missed, even if there is no "dead hand," or actual Raynaud's disease to draw special attention to them.

Then, again, many troubles of mind and its adjustment, which the authors mention, are so obviously similar in migraine and epilepsy, and after what I have elsewhere shown with regard to them, are so obviously due to uric acid as to need no comment.

As regards causation, the authors point out that epileptics come of a degenerate stock, and one subject to many kindred troubles and defects. I think, however, that much of the degeneration may be a co-result with the epilepsy rather than its cause, and we must not, I think, lose sight of the fact that not a few men whose mental powers have been distinctly above the average have none the less suffered from epilepsy, and that migraine is more common among those that work with their brains, and consequently have fairly good brain power.

As to heredity and predisposition, migraine is intensely hereditary, but the inheritance is possibly, as I have pointed out (BRAIN, Spring and Summer Number, 1893, p. 250), merely anatomical, and concerns simply the size and distribution of the arteries that supply the brain with blood, and the anatomical possibility of a good supply of blood to the brain may account for the greater frequency of migraine among brain workers.

I was much struck with this a few years ago, when a gang of eight men were moving some furniture for me, as I noticed that their foreman was a long way the youngest of the gang: but you had only to look at his face and see that he had more brain, as the saying is, than any three of the others; and it so happened that while working for me he had a typical attack of migraine, and I then found that he was an habitual sufferer, and the only one of the gang who was so.

And there is nothing extraordinary in such an anatomical inheritance, for are not eyes, noses, and teeth similarly inherited with an exactitude which is sometimes marvellous?

But in migraine, if you control the uric acid you may absolutely and entirely neglect the anatomical inheritance, which is an advantage rather than a defect.

Three generations, at least, in my own family have been affected with migraine, but in the second and third generations diet has absolutely and completely controlled it.

Epilepsy, like migraine, may be in part the result of large arteries, and therefore may occur along with considerable mental power (see Sir J. Crichton Browne, *Lancet*, 1895, ii., p. 74, also the same journal, p. 149, where a man who was very intelligent, and suffered from periodical headache and later from epilepsy, was found after death to have a heavy brain), while the eventual crippling and destruction of mind referred to by Sir J. Crichton Browne (prev. ref.) may be a result of the repeated congestion, strain and oedema, produced by the recurring high blood pressure.

It is interesting also to note that the above-mentioned author observed for himself that in "dreamy mental states,"

which he considers to be allied to epilepsy, a diet rich in animal food did harm, and he also quotes cases in which epilepsy was similarly affected by diet.

I quite agree with the authors in their argument that an accumulating poison gives the best explanation of the attacks, and I have been pointing this out for more than seven years, and indicating uric acid as the probable poison.

Then they lay great stress on the liability of women at the menstrual period, and the regularity with which attacks recur at each period; and here, again, uric acid affords a complete explanation, for nothing is simpler than to show that for some days, in every menstrual period, there is a plus excretion of uric acid ("Uric Acid," third edition, p. 188), the greatness of the fluctuation depending on the extent of the functional disturbance in the digestive and other systems, and the amount of uric acid in the body of the patient available for solution.

The same applies word for word to migraine, which bears precisely the same relation to the menstrual periods, and if the uric acid is controlled, the secondary disturbances are diminished or removed.

If the facts I have brought forward regarding the relationship between the fits and the excretion of uric acid stood alone they would be worthy of very careful consideration: but linked as they are to the similar facts about migraine and the further fact of its control and prevention by controlling the uric acid, they amount almost to a demonstration of the causation of epilepsy.

The authors divide epilepsy into (1) Reflex, and (2) Toxic; and the latter is further subdivided into (*a*) that due to Auto-intoxication; (*b*) that due to Hetero-intoxication.

Obviously, uric acid may account for toxic epilepsy of auto-intoxication, but even here a large part of the required uric acid was formed outside the body and introduced with the food, as we shall see presently.

It is, unfortunately, not in my power to study a large number of cases making similar diet alterations in all, hence I have been obliged to study one case with great care; but this plan has also some advantages of its own.

Emma S., age 20, admitted under my care at the Royal Hospital for Children and Women on December 7, 1894.

Her fits began when she was 14 years old. At present they are very regular once a week, and often on the same day each week. Her monthly periods began at 15 years of age, and are said to have been regular (but they have been absent during her stay in hospital). Her father and mother are alive and well, but her mother suffers occasionally from headache. Her eleven brothers and sisters are all in good health.

I examined the urine excreted before, during, and after a large number of her fits, and got uric acid fluctuations identical with those I had found in other cases, or in that first published (previous reference).

She was kept in bed to avoid fluctuations from muscular exertion, or changes of temperature, and then her diet and drugs were altered from time to time with a definite object and according to a pre-arranged plan, for controlling the introduction, formation, and excretion of uric acid.

It will probably suffice if I give the excretions of only one or two fits, as most of them were practically identical, and in all without exception there was a very large excretion of uric acid in the hour following the fit, having a relation of 0 to urea from 1-2 to 1-8.

During the daytime she passed water as far as possible about once every hour, so that when a fit occurred there would be but little admixture of urine in the bladder.

Then if a fit occurred, say at 3 p.m., we had got the urine passed at 2 p.m., and this was called *before*. A catheter was passed at the time of the fit, and what it brought away was called *during*, and then water was drawn or passed exactly one hour after the fit, and that was called *after*.

After always contained an excessive amount of uric acid both absolutely and relatively to urea.

Before generally contained a normal or subnormal quantity.

During sometimes contained a considerable excess, at other times only a slight excess according as the separation corresponded more or less accurately with the changes of secretion; thus, if urine was passed at 4 p.m., and if just as urine was going to be passed at 5 p.m. a fit occurred, then the 5 p.m. urine might be called *during*, but would obviously be mixed with the excretion of say three-quarters of an hour preceding the fit in which the excretion was small.

But bearing these causes of fluctuation in mind one always got

a great excess of uric acid in the hour after the fit, and a more moderate excess in the urine drawn at the time of the fit.

Thus on the day of admission she had a fit.

Before, passed at 2.15 p.m., pale straw coloured, 1009, gave—

Acidity	=	1.0 cc. of decinormal soda solution.	
Urea	=	.75 per cent.	} 1-23=1 of uric acid to 23 of urea (nearly).
Uric acid	=	.03360 per cent.	

During, drawn off at the time of the fit at 4.45 p.m., turbid, 1012, gave—

Acidity	=	1.2 cc. decinormal soda.	
Urea	=	1.0 per cent.	} = 1-17 (nearly).
Uric acid	=	.06048 per cent.	

But, obviously, there was a mixture of *during* with the urine of two and a half hours before the fit.

After.—Urine drawn at 5.45 p.m., one hour after the fit, was 60 cc., amber, turbid, 1020, and gave—

Acidity	=	7.6 cc. decinormal soda	Per Hour.	=	4.4 grs. of acidity calculated as oxalic acid.
Urea	=	1.1 per cent.	} 1-11	{	10.1 grs. urea.
Uric acid	=	.10080 per cent.			.92 grs. uric acid.

February 27, 1895.—Urine of twenty-four hours, ending this morning, was 1275 cc., turbid, 1014, and gave—

		In 24 Hours.	Per Hour.	
Acidity	4.0 cc. ..	= 49.2 grs.	= 2.0 grs.	
Urea	1.8 per cent.	= 351 grs.	= 14.0 grs.	} 1-38
Uric acid	.04704 per cent.	= 9.1 grs.	= .37 grs.	

On this day she had a fit, and the urine *during* was lost, the specimen *after* was saved, and was the excretion of exactly one hour.

It was 57 cc., amber, slightly turbid, 1022, and gave—

Acidity	8.4 cc.	=	4.6 grs.	
Urea	1.8 per cent.	=	13.8 grs.	} 1-13
Uric acid	.13440 per cent.	=	1.03 grs.	

This shows, as compared with the hourly excretion of the day before the fit, that urea was very slightly diminished after the fit, while uric acid was nearly three times as much after the fit as before it.

We see, then, that the urine, after a fit, has generally these characters: it has a high specific gravity, often very high as compared with before and during. It is often turbid, and deposits urates, or uric acid, or both. It is highly acid—this being, I think, entirely due to the large amount of uric acid (often free uric acid) which it contains, and showing nothing

whatever as to the alkalinity of the blood, which is probably high at the time of such a large excretion of urates.

Take another example, March 10, 1895.

Urine of twenty-four hours, slightly turbid, 1014.

Urea	1·6 per cent.	} 1·44
Uric acid	·03696 per cent.	

Urine, four hours before fit, 1010.

Urea	·9 per cent.	} 1·34
Uric acid	·02688 per cent.	

Urine drawn at time of fit, containing, of course, also the excretion of the previous four hours, 1014.

Acidity	·8 cc.			
Urea	1·2 per cent.	} 1·40
Uric acid	·03024 per cent.	

Urine, one hour after the fit, pale amber, turbid, 1022.

Acidity	5·6 cc.			
Urea	1·0 per cent.	} 1·20
Uric acid	·05040 per cent.	

Here we have a diminished excretion, 1·44, in the twenty-four hours preceding the fit, a slightly increased excretion, 1·34, in the urine passed four hours before the fit.

A diminished excretion in the urine drawn at the time of the fit, being also the excretion of the four hours preceding it, 1·40, showing that there was a fluctuation in the direction of minus excretion in the hours immediately preceding the fit.

And, lastly, we have a plus excretion in the hour following the fit—this being, I think, probably composed of a very large excretion in the 20-30 minutes following the fit, with a lessened excretion after this, the differences in the suddenness and steepness of the waves of fluctuation accounting for the differences in relation found after different fits.

With regard to the large excretion *after* the fit, we must bear in mind that it is the uric acid in the blood that alters the blood pressure, and affects the intracranial circulation; and it is only after it has been in excess in the blood for some little time, that it begins to be found in excess in the urine.

As regards effects on the blood pressure and intracranial circulation, these are probably most marked just at the time of change from low to high pressure; hence the fit comes early in the uricacidæmia, and precedes the great excretion of uric acid.

See, again, March 19, 1895, urine of twenty-four hours, 1·24.

Urine at 9.15 a.m., 1·56.

Urine at 12.15 p.m., time of fit, 1-70.

Urine after fit, 1.15 p.m., 1-9.

Here we have a rather plus excretion on the day before the fit, a considerable retention before 9 a.m., a greater retention between 9 and 12 followed by a very greatly plus excretion between 12 and 1 p.m.

Evidently the fluctuation which produced this fit began about 7 or 8 a.m., and the retention continued and increased up to about 12 o'clock noon, then followed the opposite and resulting plus excretion, producing the fit at 12.15 and the excretion of 1-9 between that and 1.15 p.m.

The parallel between this fluctuation and that which produces the uric acid headache is almost absolute, the differences are those of time and degree only.

Now we can control the uric acid headache by controlling the uric acid, to an extent which leaves little to be desired; can we control also uric acid epilepsy by the same means?

This was the question I have endeavoured to answer by observing this case, and I chose it in preference to other cases because the history seemed to show that the fits were here remarkably regular in their weekly cycle.

I examined the urine *before, during and after* more than fifteen of her fits, and the results in all were as regards the main points practically identical with those given, and I would undertake to tell in any case when urine was collected in this manner which was the urine of the hour following the fit. This urine also has a characteristic which I have not mentioned, though it is of very great importance, viz., that when a patient is taking drugs such as an iodide or a salicylate, which give a marked reaction in the urine, such reaction is almost completely absent from the urine passed in the hour following the fit.

I fortunately noticed this point in the urine of the very first case of epilepsy I examined, and I have mentioned it elsewhere¹ as evidence of the very great effects which excess of uric acid in the blood exerts on the circulation of the stomach and intestines, more or less completely suspending digestion and absorption in these viscera; thus explaining also both the oxaluria of paroxysmal hæmoglobinuria² and dyspepsia, and the presence of excess of putrefactive products from the intestines in the urine after an epileptic fit. Under these conditions, not only is there no absorption of drugs from the stomach and intestines, but putre-

¹ "Uric Acid," third edition, p. 206.

² *Ibid.*, p. 390.

faction soon takes the place of digestion, and the products formed under its influence account completely for the oxalates and excess of æthereal sulphates found later on in the urine.

To suggest, as some have ventured to do, that these putrefactive products found in the urine after a fit may represent the cause of the fit, is to put the cart in front of the horse, make a hopeless muddle of cause and effect, and bring a pea soup fog over a clear sky. The only simple, complete, and efficient explanation is, that the circulatory changes which excess of uric acid in the blood produces, affect, on the one hand, the intracranial circulation and produce the fit, and on the other, the stomach and intestines and bring digestion and absorption to a standstill; failure of absorption explains the absence of the drugs from the urine, and excess of putrefaction explains the production of the products which ultimately appear there. Precisely the same thing also occurs in the uric acid headache, for when this is severe, drugs introduced into the stomach produce absolutely no physiological effect, and do not appear in the urine till the headache is passing off, and if the gastric upset is continued for some time, it often ends in vomiting, and the food is then found to be almost undigested, though it has, perhaps, been in the stomach long enough to have been completely absorbed.

In all these cases if you control the uric acid you will control also the headache, the fits, and the gastric upset—all will improve together, for all are due to one and the same cause, the altered circulation which is common to the whole body.

Now, in the case of Emma S., on December 30, 1894, she had a fit while taking both salicylate of soda and iodide of potassium three times a day, the urine *before* the fit gave lots of iodide when a silver salt was added, and a dark purple with perchloride of iron.

Urine during gave distinctly less iodide.

Urine after with a relation of uric acid to urea of 1-10, gave no iodide of silver, and only a very slight purple with the iron.

To save space I shall now give the whole of her fits in the form of a table, in which I shall also note the interval between each fit and the one preceding it, health, diet, drugs, and remarks; and we can thus see at a glance any variations presented by the fits and the corresponding variations in diet and drugs made with the object of affecting uric acid.

No. of Fits.	Date.	Interval.	Health.	Diet.	Drugs.	Remarks.
	1894.					
1	Dec. 7	7 days	Average	Ordinary	None	Soon after admission
1	" 19	12 "	"	Milk only	Iodide of mercury, Dec. 10	3 in 30
1	" 30	11 "	"	" "	20th, Salicylate of soda, and iod. potssm.	
	1895.					
1	Jan. 6	7 "	Tonsillitis T. normal on 4th	" "	Salicylate of soda alone	3 in 32
1	" 18	12 "	Average	" "	Salicylate of soda off, 12th	
	" 21	...	"	Milk & fish	None	
	" 24	...	"	Milk, fish & meat	"	
1	" 31	13 "	"	Milk, fish & meat	"	
1	Feb. 4	4 "	"	Milk, fish & meat	None	3 in 17
1	" 11	7 "	"	Milk, fish & meat; beef tea added	"	
1	" 17	6 "	"	16th, beef tea off	"	
	" 21	Meat off		
	" 25	Fish off		
1	" 27	10 "	"	Milk only	None	3 in 30
1	Mar. 10	11 "	"	" "	"	
	" 11	...	"	Milk & beef tea	"	
1	" 19	9 "	"	Milk only; last beef tea on 16th	"	
	" 21	Tea		
2	" 25	6 "	"	Farinaceous, with 4 cups tea=2-3 grs. theine p. dy.	...	3 in 18
	" 29	...	T. raised slightly	
1	Apl. 2	8 "	Average	"	None	
1	" 6	4 "	"	"	"	3 in 29 ¹
	" 11	...	"	Farinaceous, no tea	"	
1	" 17	11 "	"	" "	None	
1	" 24	7 "	"	" "	"	
1	May 5	11 "	"	" "	"	
	" 6	...	"	Farinaceous, & tea again	None	3 in 26
1	" 13	8 "	"	
1	" 21	8 "	"	"	"	
1	" 31	10 "	"	"	"	

¹ The previous 3 in 18 was probably partly the result of the beef tea.

Her first fit was on December 7, said to be one week after the previous one; her health and diet were those of her ordinary life, and she was taking no drugs.

On December 10 I put her on absolute milk diet, and gave her a mixture containing perchloride of mercury and iodide of potassium.

On December 19 she had a fit, twelve days after the previous one.

On December 20 I stopped the mercury, and gave iodide of potassium and salicylate of soda—leaving the milk diet as before.

On December 30 she had a fit, eleven days after the previous one—making three fits in thirty days.

On January 2, 1895, she got slight tonsillitis, with raised temperature, which became normal on the 4th.

On January 6 she had a fit, seven days after the previous one, but I think that this short interval was probably in part the result of the attack of tonsillitis, which would cause a fluctuation in urate excretion.

After the fit on January 6, the iodide was left off, and the salicylate given alone, and all drugs were left off on January 12.

I now began to test the effects of diet.

On January 18 she had a fit, twelve days after the previous one, while she was still on milk only.

On January 21 fish was added to her diet, and on the 24th meat was added, and she continued to take milk, fish, and meat till February 11, when beef tea was added, and she continued to take milk, fish, meat, and beef tea till February 16, when the beef tea was taken off; on the 21st the meat was taken off, and on the 25th fish was taken off, and she was again on milk alone.

She had a fit on January 31, thirteen days after the previous one, and making three fits in thirty-two days. At the date of the last of these fits she had been on fish for ten days and on meat for seven days.

So far, then, diet had not affected the frequency of the fits, for thirteen days was the longest interval she had had; indeed, it rather seemed as if the addition of flesh food had increased the interval, and I should not be surprised if this was the case, for, in the case of the uric acid headache, taking meat steadily every day does not produce a headache nearly so quickly as taking it only for one day or one meal; and the reason is, that the steady daily introduction of uric acid tends at first to keep the blood clear, while a single dose does not raise the acidity much, and as soon as the acidity falls it gets into the blood, and produces signs of its presence.

The next fit was on February 4, four days after the previous one, and the next on February 11, seven days after that, and the next on February 17, six days later, making three fits in seventeen days—a very different record from the two previous series.

From February 25 to March 11 she was on milk only; on March 11 beef tea was added and continued till March 16.

On February 27 she had a fit, ten days after the previous one, a second on March 10, eleven days after, it being on milk only, and a third on March 19, nine days later, when she had been on milk and beef tea for eight days: this making three fits in thirty days, or the same record as the first two series.

Now came what was rather a surprise to me: on March 16 the beef tea was left off, and on March 21 she was put on farinaceous diet, which includes bread, puddings, and tea, in addition to milk.

She now had a fit on March 25 (or rather two close together, which I count as one, as both were due to the same fluctuation of urates), six days after the previous one; another on April 2, eight days later, and a third on April 6, four days after that, making three fits in eighteen days.

Now, as I say, this was rather a surprise, for though I expected that the beef tea might have some effect, it was left off on March 16, and yet the quickest time of all was the fit on April 6, four days after the previous one.

I confess that at this point I felt rather at sea: but I chanced one afternoon to go into the ward just as she had finished her tea, and looking into her cup I saw that what remained was very dark coloured, and it had evidently been rather strong. This led me to enquire how much tea was included in the farinaceous diet, and I found that she had four breakfast cups a day, two at breakfast and two at tea, and further enquiring as to the amount of tea leaves used to make this amount of tea, and taking it that one pound of tea contains 175 grains of theine, I found that she was thus introducing into her body nearly 3 grains of theine a day.

And as I can show that caffeine, theine, theobromine, and all similar xanthin compounds, are practically equivalent to uric acid, she was thus quietly pouring in the equivalent of at least 20 grs. of uric acid a week.

And it was thus little to be wondered at, that with the help of the beef-tea from March 11 to 16, her fits had been more frequent.

After making this discovery I stopped her tea on April 11, leaving her farinaceous diet otherwise as before.

She now had a fit on April 17, eleven days after the previous one, another on April 24, seven days later, and a third on May 5, eleven days after that, making three fits in twenty-nine days.

On May 6 she was again given tea, her diet being otherwise unaltered, and she had a fit on May 13, eight days after the previous one, a second on May 21, eight days later, and a third on May 31, ten days after that; making three fits in twenty-six days, or slightly quicker than in the previous series.

I now decided that I would try and diminish the frequency of the fits, so I put her on farinaceous diet without tea, and gave her salicylate of sodium with iodide of potassium, the former to eliminate as much as possible of the uric acid she might have in her body, and the second to prevent it from combining with alkali when in the blood: for it is apparently the combination of uric acid with alkali in the blood which produces all the physiological and pathological effects which I have been led to attribute to uric acid. I also allowed her to get up, she having been in bed the whole of the previous period, about six months in all.

With this change of diet and drugs she went without a fit for a whole month, and she had no further attack when she left the hospital at the end of June.

The diet treatment did not cause any loss of weight, as in a case I have elsewhere mentioned,¹ for she kept about 90-91 lbs. during the whole of her stay in the hospital.

And now for a general survey of the fluctuations shown in the table.

The fits, according to history, were very regular—one in a week, or three in twenty-one days. After admission on milk only and some drugs, they fell to three in thirty and three in thirty-two days; then animal food was put on, and they quickened up to three in seventeen days; this was left off again and they fell to three in thirty days; some beef tea and tea were given and they quickened to three in eighteen days; these were left off and they fell to three in twenty-nine days; tea was put on again and they quickened slightly to three in twenty-six days.

And then when tea was left off and some drugs were given

¹ "Uric Acid," third edition, p. 209.

as at first they fell lower than they had been during the whole of her previous stay in the hospital, thirty days elapsing without any fit.

There are no doubt some who would argue that this was all pure chance, and that her fits would have varied in much the same way if no alterations in diet or drugs had been made.

I can only say that such an argument has little in it to recommend itself to my mind; for it almost passes the bounds of possible chance that the fits should by accident have varied in such remarkable correspondence with the changes we made in the uric acid introduction (see "Uric Acid," figs. 23-28 and 43-47).

It leaves out of count also entirely the numerous cases in which a diet free from flesh food has produced a more or less marked alteration in the number of fits,¹ or a diet in which the flesh food is only reduced has produced a distinct reduction.²

I have seen it suggested that meat food acts as a general stimulant to the nerve centres, like corn in the food of horses; but in the uric acid headache (migraine) this is certainly not the case, as this headache is not influenced at all by the general level of nitrogenous excretion, but only by the height of uric acid above urea, which is a measure of the quantity of uric acid passing through the blood; and this headache can be produced by the ingestion of uric acid or xanthin compounds equivalent to it, which increase the excretion of uric acid but hardly affect total nitrogen; and in the case narrated in this paper it rather appears as if beef-tea and tea, which introduced xanthin and uric acid, had a similar effect on the fits of epilepsy.

On the other hand, in migraine you may keep urea well above the physiological level ($3\frac{1}{2}$ grs. per pound of body weight per day), and provided you do this with milk and vegetable substances which do not introduce much uric acid, there will be no increase of headache.

The point about migraine beyond which there is no appeal

¹ "Uric Acid," third edition, p. 176; *Lancet*, 1895, vol. i., p. 96.

² *British Medical Journal*, 1895, vol. i., pp. 1088 and 1420.

is, that if you control uric acid, you control the headache without attending to anything else, and I am now suggesting that the same will be found to hold for epilepsy, only here the control must be more powerful and complete.

It further takes no notice of the fact that it is almost impossible to regard the uric acid fluctuation above described as a consequence of the fits, while it is easy, looking to the important influence which this substance exerts on the nutrition and function of almost all the tissues of the body in the uric acid headache, for instance, to regard it as a cause.

Then I believe that as a matter of fact there are really to be met with all gradations from a mere temporary absence of mind up to a severe epileptic fit, and nothing is more common than slight absence of mind in mental depression, or the uric acid headache (see also Sir J. Crichton Browne, *Lancet*, 1895, vol. ii., p. 4, for many interesting connections between dreamy mental states, migraine, and epilepsy).

In the uric acid headache reasoning is difficult, and has to be forced, and the mind becomes a complete blank for words or for names, which in ordinary conditions one knows almost as well as one's own; on the other hand, either before or after the headache, when the blood is cleared of uric acid, there is mental brightness and well-being, a feeling of pleasure in living and thinking, and work is done at double speed and with comparatively little trouble; and all these things I can answer for it, vary absolutely with the uric acid in the blood.

And not only so, but the nutrition and secretion of every gland in the body, the activity of the muscles, and even the formation of urea, are absolutely controlled and conditioned by the influence of uric acid on the circulation, and this having been proved it is obviously quite within the power of this substance to produce such changes in the cerebral circulation as will amply account for the fits of epilepsy.

I shall not further argue the point, but desire to leave this case to the judgment of the profession; and I have a further object in publishing it, namely, to draw the attention of fellow-workers to the fact that they must give far more

care and attention to the cutting off of all possible introduction of uric acid than they have hitherto done, if they are to succeed in curbing the fits of epilepsy.

It is no use to reduce flesh food to one-half or one-quarter of previous quantities, leaving soup, tea, coffee, cocoa, &c., to be taken as before. Why, these measures would not even prevent the uric acid headache; and epilepsy, as I have shown, is probably a more difficult matter to deal with. Here flesh food must be avoided entirely, and all soups and meat extracts regarded as poisons; while tea, coffee, and cocoa, and all other vegetable substances containing xanthin compounds, are to be regarded as containing uric acid, and used, if at all, only as the merest flavouring.

With such diet alterations I believe that much may be done for many cases of epilepsy: but much care is necessary as it is useless to close one door and leave another open, as I found myself doing in the case of tea; and this may explain my comparatively small success with diet in some previous cases, as I have only recently found out that the above xanthin compounds are equivalent to uric acid.

Meanwhile the facts mentioned above show pretty clearly that the fits of epilepsy and convulsions in general bear an extremely close relationship to the uric acid headache (migraine), and like this are probably functional disorders, due to altered circulation in the brain,¹ such altered circulation again being due to the contracted arterioles, and high blood pressure, which I have shown that uric acid produces.² The headache is controlled with almost absolute certainty by a diet which frees the blood from excess of uric acid. I trust we shall yet find that many fits will yield in the same way to well directed treatment.

¹ BRAIN, Spring and Summer Number, 1893, and BRAIN, Spring Number, 1891.

² "Uric Acid," third edition, chapter v.

JAMES BRAID, SURGEON AND HYPNOTIST.

BY DR. J. MILNE BRAMWELL.

JAMES BRAID, the subject of this sketch, was born at Ryllaw House, in Fifeshire, about 1795. He studied at Edinburgh University and, after qualifying as a surgeon, practised for a short time in Scotland; then removed to Manchester, where he remained up to the time of his death.

On November 13, 1841, Braid, for the first time, was present at a mesmeric *séance*, the operator being Lafontaine. At this time mesmeric phenomena were believed to be due either to some mysterious force or fluid, self-deception, or trickery. Braid held the latter theory and, on the first occasion, saw nothing to cause him to alter his views. At the next *séance*, six days later, he noticed that one subject was unable to open his eyes. Braid regarded this as a real phenomenon, and was anxious to discover its physiological cause; and the following evening, when the same case was again operated on, he believed he had done so. After making a series of experiments, chiefly on personal friends and relations, he expressed his conviction that the phenomena he had witnessed were purely subjective, and began almost immediately to place these views before the public, his first lecture being delivered on December 27, 1841.

In 1842 Braid offered a paper on the subject of hypnotism to the Medical Section of the British Association. This was refused; whereupon he gave a *Conversazione*, at which many members of the Association were present, read his paper, and showed cases. His first work on mesmerism was

entitled "Satanic Agency and Mesmerism reviewed, in a Letter to the Rev. H. McNeile, A.M., of Liverpool, in reply to a Sermon preached by him at St. Jude's Church, Liverpool, on Sunday, April 10, 1842." McNeile had charged Braid with "refusing to state the laws of nature by the uniform action of which mesmeric phenomena were produced." To this Braid replied, that he had always explained the phenomena on physiological and psychological principles, and that McNeile had persistently refused to attend his lectures, or read any account of them.

"Neurypnology, or the Rationale of Nervous Sleep," published by Braid in 1843, and of which 800 copies were sold in a few months, was the first of a series of works on the subject of hypnotism. The necessarily limited length of this article does not permit me to deal with each of these separately and in detail. I propose, therefore, first to give a short account of the theories which appear in "Neurypnology" alone, and then to group together the more important of the facts and views found in the other works.

Neurypnology.—At the second *séance* Braid observed that the mesmeric condition was induced by fixed staring, and concluded that the inability to open the eyes arose from paralysis of certain nerve centres and exhaustion of the levator muscles. "I expressed," he said, "my entire conviction that the phenomena of mesmerism were accounted for on the principle of the derangement of the state of the cerebral spinal centres, and of the circulatory, respiratory, and muscular systems, induced by a fixed stare, absolute repose of body, fixed attention and suppressed respiration, concomitant with that fixity of attention. That the whole depended on the physical and psychical condition of the patient arising from the causes referred to, and not at all on the volition or passes of the operator, throwing out magnetic fluid, or exciting to activity some mystical, universal fluid or medium." Braid not only insisted that the condition was a purely subjective one, produced in this mechanical way, but he also claimed to have successfully demonstrated that it could be induced in like manner in persons who had never heard of mesmerism, and who were

ignorant of what was expected of them. In illustration of this, he mentioned that he had hypnotised one of his servants, who knew nothing of mesmerism, by giving him such directions as were calculated to impress his mind with the idea that his fixed attention was required merely for the purpose of watching a chemical experiment with which he was already familiar.

After having established the subjective nature of the phenomena, Braid suggested that these should be called hypnotic instead of mesmeric, and invented the terms hypnotism, hypnotist, hypnotise, &c., which, with little change, still form to-day the recognised terminology of the subject.

Braid induced hypnosis by making the subject look at a bright object, held at such a position above the forehead as was calculated to produce the greatest possible strain upon the eyes and eyelids, while at the same time the mind was to be riveted on the idea of that one object. After hypnotising his patients, he manipulated them in various ways, in order to cause changes in the muscular and circulatory systems; he believed that this excited the different hypnotic phenomena, and played an important part in the cure of disease.

Braid found that he could terminate the hypnotic condition by means of a current of cold air. He also noticed that he could make a rigid limb flexible by blowing on it; that he could restore sight to one eye by the same means, and leave the other insensible; excite one half of the body to action while the other remained rigid and torpid, or make the patient pass from a general state of inactivity of the organs of special sense and tonic muscular rigidity to the opposite condition of extreme mobility and excited sensibility. He acknowledged that he was unable to explain these extraordinary phenomena, but stated that he had no difficulty in reproducing them, that they were independent of any "*rapport*" between operator and patient, and invariably appeared no matter whether the current of air came from the lips, a pair of bellows, the motion of the hand, or any inanimate object.

The subjective explanation of the origin of mesmeric phenomena was not a new one, and had already been given both by the Abbé Faria and Bertrand. Their views, however, if not entirely forgotten, exercised no practical influence on mesmeric theory, and Braid evidently was unacquainted with them when he commenced his mesmeric researches; thus, his conclusions were arrived at independently, and successfully substituted for those universally held in his day. At a later date, when his opponents pointed out the similarity between the theories, Braid asserted that this was more apparent than real, as Faria had attributed everything to the effect of imagination; on this point they differed, but were alike in asserting that neither contact nor magnetic fluid was necessary.

At this time Braid did not believe that the phenomena of hypnotism were the result of attention, for, in speaking of some articles on animal magnetism which had appeared in the *Medical Gazette* in 1833, he said: "In the writer's opinion the phenomena are the result of attention strongly directed to different parts of the body, whereas, by my method the attention is riveted to something outside the body."

In opposition to the theory that hypnotism resembled reverie, Braid said: "Reverie proceeds from an unusual quiescence of the brain, and inability of the mind to direct itself strongly to any one point. There is defect in the attention, which, instead of being fixed on one subject, wanders over a thousand, and even on these is feebly and ineffectively directed. This is the very reverse of what is induced by my plan, because I rivet the attention to one idea, and the eyes to one point, as the primary and imperative condition."

Some experiments Braid made, as to the methods of inducing hypnosis, appear to have first suggested alterations in his hypnotic theory and shaken his faith in the purely physical explanation of hypnotic phenomena. At first he had required his patients to look for a considerable time at some inanimate object until the eyelids closed involuntarily. He frequently found, however, that this was followed by pain and slight conjunctivitis, and in order to avoid this he closed the patient's eyes at a much earlier stage. Despite this he

was able to hypnotise as easily as before and without subsequent unpleasant sensations. This led to further experiment, when he found he could induce hypnosis as readily in the dark or with the eyes bandaged, as in the light and with the eyes uncovered; it being only necessary to keep the eyes fixed and the body and mind at absolute rest. He always failed, however, in young children and in persons of weak intellect, or of restless and excitable minds, who were unable to comply with these simple rules. As he succeeded with the blind, Braid concluded that the impression was made through the mind and not through the optic nerve.

“It is important to remark,” he said, “that the oftener patients are hypnotised, from association of ideas and habits, the more susceptible they become; and in this way they are liable to be affected entirely through the imagination. Thus, if they consider or imagine there is something doing, although they do not see it, from which they are to be affected, they will become affected; but on the contrary, the most expert hypnotist in the world may exert all his efforts in vain, if the party does not expect it, and mentally and bodily comply, and thus yield to it.”

Braid at first was inclined to believe in phrenology, and thought it possible that the passions, emotions, and intellectual faculties could be excited during hypnosis by simple contact or friction over certain sympathetic points of the head and face. He cited twelve cases in which he believed he had observed these phenomena, and thought that they appeared in subjects who were ignorant of phrenology, and who were not influenced by previous training, or by leading questions and suggestions on the part of the operator. He was not satisfied with these results, however, and stated that it was his intention to conduct a new series of experiments on fresh patients, in order to ascertain to what extent it might be practicable, by arbitrary association, to excite the opposite tendencies from the same point. He also thought it probable that errors might have arisen through the remarkable docility of hypnotic subjects, which made them anxious to comply with every suggestion or indication given by the operator.

Later Works.—Braid recognised two distinct hypnotic conditions, which correspond practically with the late Mr. Edmund Gurney's "alert" and "deep" stages. The deep stage was characterised by a condition of torpor more profound than that of natural sleep; the alert by exaltation of the special senses and increase in muscular sense and power, as well as exaltation of certain mental faculties. There were intermediate stages between the two, and frequently one glided imperceptibly into the other. By the sense of smell some persons were able to detect any person known to them, or to find the owner of any glove. They first smelt the glove, and then unhesitatingly presented it to its owner, choosing him from amongst a large company. If the nostrils were stopped, however, the apparent clairvoyant faculty instantly disappeared. The sense of touch and the muscular sense were also sometimes so remarkably increased, as to enable certain subjects to write with great accuracy during hypnosis, when effectual precautions were taken to prevent their being able to see. They crossed the "t's" and dotted the "i's," and could even go back a line, strike out a letter and put it in its proper place. One patient could correct the writing on a whole page of notepaper, but if the relative positions of the paper and table were changed, the alterations ceased to be placed correctly. A full and remarkable account is given of the power possessed by two somnambules of imitating songs, &c., in different languages. One of them was severely tested by several experts, particularly by Jenny Lind, and accompanied her successfully when she sang Swedish and Italian songs, and executed complicated and difficult exercises. This patient was ignorant of the grammar of her own language when awake, and possessed no unusual musical gifts.

Braid found that hypnotism gave him the power of directing or concentrating nervous energy; of exciting the force and frequency of the heart's action and regulating the state of the general and capillary circulation; of influencing the muscular tone and energy, as well as the whole of the secretions and excretions of the body. He could also prevent the pain of operations, and cure a number of

diseases which ordinary treatment had failed to relieve—in fact, excite a variety of phenomena widely different from those occurring in natural sleep, or during the waking condition.

Braid noticed that hypnotised patients, who remembered nothing on awakening, even after having undergone surgical operations, could recall what had passed on being re-hypnotised. He termed this “double consciousness.” The operator could also arouse the memory of the hypnotised subject by placing his hand on any part of his body; this was simply a physical aid to the concentration of attention. Braid taught some of his somnambules Greek, Latin, French, and Italian, which they forgot on awaking, but remembered on being re-hypnotised. He was aware of the fact that many hypnotic phenomena could be produced without the subjects having previously passed through a condition in any way resembling sleep. This was described as the electro-biological state—a name which Braid justly termed ridiculous. Despite long perseverance, he never succeeded in hypnotising idiots, and found that one patient who was easily hypnotised in health, became refractory during the delirium of fever. Hypnotism, he thought, explained the prolonged trance of the fakirs and the voluntary suspended animation of Colonel Townsend.

Braid performed a number of minor operations on hypnotised patients without their experiencing any pain; in some of these cases the hypnosis was slight, and there was no interference with the general consciousness or memory. Relief or cure was obtained by hypnotic treatment in the following diseases:—Painful affections of muscles and joints persisting after injury, curvature of the spine, corneal opacity, ophthalmia, heart disease and dropsy, organic paralysis, certain skin diseases, amenorrhœa, leucorrhœa, rheumatism, gout, dyspepsia, constipation, certain forms of deafness, epilepsy, neuralgia, nervous headaches, somnambulism, catalepsy, monomania, *idée fixe*, delirium tremens, torticollis, and various other forms of muscular spasm, chorea, hysterical paralysis, and aphonia.

In a case of spinal curvature, which had been treated by

ordinary methods for six years without benefit, Braid obtained marked and speedy improvement by removing all mechanical apparatus, and rendering the morbidly weak muscles cataleptic by suggestion. He believed he could cure recent cases in this manner without pain or inconvenience to the patient, and considered mechanical supports—except where there was actual disease of the bone—worse than useless, as they favoured the weakening of the muscles.

In 1845, Braid cited the case of a girl, aged 14, who suffered from various hysterical symptoms and frequent attacks of spontaneous somnambulism and catalepsy; he succeeded in putting himself *en rapport* with her during an attack, and cured her by hypnotic treatment. One day he found her suffering from constipation, and successfully suggested during hypnosis that her bowels should act in five minutes. He mentions this as the first instance he knows of in which action of the bowels was produced by suggestion, and afterwards he was equally successful with other patients.

One of the most remarkable cases was that of a near relative of Braid's, who suffered from rheumatism and from long-standing and extensive opacity of the cornea. Braid hypnotised her for the former affection, when, to his astonishment, the cornea gradually became transparent, and ultimately it required close inspection to detect any opacity.

According to Braid, the hypnotic method was superior to all others for regulating the catamenial functions, and for curing or relieving morbid conditions connected with them. His most brilliant results, however, were obtained in hysterical paralysis, and in many of these all ordinary methods of treatment had entirely failed. He records an interesting case in which he succeeded by suggestion in augmenting and maintaining the secretion of milk. On more than one occasion, Braid succeeded in hypnotising himself, and in obtaining relief from rheumatic pains.

Theory.—After referring to the reasons which had induced him to adopt the word hypnotism instead of that of mesmerism, Braid stated that he had come to the conclusion that further alteration in the terminology was necessary.

Under hypnotism had been comprised a series of widely varying stages or conditions, whereas, he thought it ought to be restricted to that state resembling sleep followed on awakening by complete forgetfulness of what had occurred. Of those who were relieved or cured by hypnotism only one in ten reached this stage, and the remainder were apt to imagine they could not be benefited by processes which failed to produce the phenomena the name implied. With the exception of the condition of sleep, just referred to, followed by amnesia, Braid now proposed to substitute the term *monoideism* for that of *hynotism*, *monoideise* for *hypnotise*, &c., for the following reasons :—No matter in what way the hypnotic state was induced, it was essentially one in which the mind became so completely concentrated upon a single idea or train of thought as, for the time being, to make it dead or indifferent to all other considerations and influences. The various physical phenomena were the result of this mental condition, and were simply exaggerations of what had been observed in the ordinary waking state, when the attention had been fixed on one part or function of the body, and withdrawn from others. The imagination, faith and expectant ideas of the hypnotised subject rendered the influence of attention more powerful than in the ordinary waking condition. The mental and physical phenomena, by whatever processes induced, resulted entirely from dominant ideas in the minds of the subjects. Whether these ideas existed previously, or were induced by the audible suggestions or sensible impressions of the operator, before or after hypnosis, was absolutely immaterial. This theory was first published by Braid in 1847. The fascination of birds by serpents, the phenomena of electro-biology, of table-turning, the gyrations of the odometer of Dr. Mayo, the magnetometer of Mr. Rutter, the movements of the divining rod, the supposed levity of the human body when lifted on the tips of the fingers of four individuals were all, according to Braid, examples of unconscious or involuntary muscular action resulting from dominant ideas. When the attention of man or animal, he said, is absorbed by an idea associated with movement, a current of nervous force is sent into the

muscles and a corresponding motion produced, not only without conscious effort, but even, in many instances, in opposition to the volition. The subject loses the power of neutralising the dominant idea, and is irresistably drawn or spellbound according to the nature of the impression produced and may, in this way, be brought under the control of others by means of audible, visible and tangible suggestions.

In "Neurypnology," Braid stated that he was unable to account for the action of a current of air upon hypnotised subjects; at a later date he gave the following theory. In hypnosis the attention is concentrated upon the particular function called into action, while the others merge into a state of torpor; thus, only one function is active at any one time, and hence intensely so. The arousing of any dormant function is equivalent to superseding the one in action, and he explained, therefore, the termination of a state of muscular rigidity, when a current of cold air was directed to the skin, by suggesting that this called attention to the skin, and withdrew it from the muscular sense.

Braid regarded the following as the chief points of difference between spontaneous and induced somnambulism. Spontaneous somnambulists are impelled to certain trains of action by internal impulses, while the induced tend to remain at absolute rest, and to lapse into a state of profound sleep, unless excited by some impression from without.

Natural and artificial sleep were not regarded by Braid as identical, and the following are further points of contrast in addition to those already mentioned. The pupils are dilated in hypnosis, and the eyelids either quiver continuously, or are firmly closed by spasmodic contractions. In passing into hypnosis, anything held in the hand is grasped still more firmly. There is an increase of the muscular sense, and hypnotic subjects possess an extraordinary power of balancing themselves. Chronic rigidity of the muscles is not followed by corresponding exhaustion. Sometimes, in the hypnotic state, there is excitement, similar to that produced by wine and spirits; at others, there is muscular quiescence, with acute hearing and dreamy, glowing imagination—closely resembling the con-

dition induced by conium. While falling into natural sleep, the mind is diffusive or passive, flitting indifferently from one idea to another, and the subject is unable to fix his attention or to perform any act requiring much effort of will. This state is carried into the sleep, and audible suggestions or sensible impressions, if not intense enough to wake the sleeper, seldom do more than arouse dreams, in which ideas pass through the mind without exciting definite physical acts. On the other hand, the active and concentrated state of mind created by the induction of hypnosis is maintained, and the subject manifests various physical acts in response to direct or indirect suggestion. Another difference is the wonderful power of hypnosis in curing many diseases, which have resisted natural sleep and every known agency for years. For instance, a few hypnotic *séances* of ten minutes each cured a patient who must have had, during his long illness, at least eight years' sleep.

Magnets, &c.—In Braid's time, the mesmerists held that magnets, certain metals, crystals, &c., possessed a peculiar power, and, with sensitive subjects, were capable of producing attraction and other remarkable phenomena. Some experienced an unpleasant sensation like an aura; others got headache, attacks of fainting, or catalepsy, with spasms so violent that they apparently endangered life. Frequently there was hyperæsthesia of the special senses, and many saw fiery bundles of light stream from the poles of the magnet. All this was said to happen when the subjects did not see the magnets, and did not know what was being done. Braid performed many experiments in order to test these statements, with the following results:—The phenomena appeared when the patients had preconceived ideas on the subject, or when these were excited by leading questions; but were invariably absent when they were ignorant of what was being done. Pretended magnets also produced the phenomena when the patients knew what was expected to occur. Reichenbach recorded an instance where, by the mere exposure of a sensitive plate in a box with a magnet, an impression had been made, as if it had been exposed to the full influence of light. Braid repeated

the experiment, and also had similar ones performed for him by an expert photographer, and, when all sources of fallacy were guarded against, the results were invariably negative. According to Braid, the mind of the patient alone was sufficient to produce the effects attributed to magnetic or odyllic force, and suggested ideas were capable of exciting a great variety of physical sensations and mental conditions. In 1843, Braid referred to Elliotson's belief in the powers of certain metals, and to Wakley's experiments. The latter, operating with a non-mesmerising metal, made the patient believe he was using a mesmerising one, whereupon she fell asleep; and he concluded that all the subjects were impostors. Braid denied this, asserting that the active agent was simply the imagination, and that the metals were neither mesmeric nor non-mesmeric. In the same way, he explained the action of the wooden tractors which Dr. Haygarth successfully substituted for the metal ones of Mr. Perkins.

As the result of some experiments performed in America, it was stated that certain medicines could manifest their influence through glass. Braid repeated these, and found the result invariably a negative one, when mental influences were excluded. He was able, however, to make a phial filled with coloured water act as an emetic when he confidently predicted this; while a similar phial, placed in the patient's other hand, checked the emetic action when he asserted that it would do so.

By the action of the imagination, Braid explained not only the curative power of homœopathic remedies, but also that of some which had a recognised place in orthodox medicine. Every drug had a two-fold influence; first, that due to its physical properties alone; and secondly, that which resulted from the patient's mental expectation of predicted effects. The influence of the mental attitude of both physician and patient caused remarkable revolutions in the estimate of the powers of particular medicines. At one time a universal favourite apparently possessed every valuable quality; at another, was discarded as utterly worthless, to be again taken into favour at a later date.

Clairvoyance, &c.—In Braid's opinion, much of the reluctance to accept the genuine phenomena of mesmerism and hypnotism arose from the extravagant assertions of the mesmerists in reference to clairvoyance—assertions which he described as being opposed to all the known laws of physical science, and a mockery of the human understanding. As the result of frequent experiments made, not only on his own patients, but also upon many of the renowned clairvoyants of the day, Braid was able to find nothing but hypnotic exaggeration of natural powers. The following sources of error, he said, ought always to be kept in mind:—(1) The hyperæsthesia of the organs of special sense, which enables impressions to be perceived through the ordinary media that would pass unrecognised in the waking condition. (2) The docility and sympathy of the subjects, which tend to make them imitate the action of others. (3) The extraordinary revival of memory by which they can recall things long forgotten in the waking state. (4) The remarkable effect of contact in arousing memory. (5) The condition of double consciousness or double personality. (6) The vivid state of the imagination, which instantly invests every suggested idea, or remembrance of past impressions, with the attributes of present realities. (7) The tendency of the human mind, in those with a great love of the marvellous, to erroneously interpret the subject's replies in accordance with their own desires. (8) Deductions rapidly drawn by the subject from unintentional suggestions given by the operator.

Braid considered that the belief in thought transference arose from the failure to guard against sources of error, similar to those just described, and stated that he had never met with any case where the subjects could correctly interpret his unexpressed desires, without some sensible indication of them.

In reference to the alleged intuitive powers of certain mesmeric subjects, Braid stated that, whereas with animals instinct was usually right, with somnambules it was generally wrong. It was true that certain patients could successfully predict their own hysterical attacks, but here

the prophecy produced its own fulfilment through auto-suggestion.

By 1843 Braid had entirely abandoned all belief in phrenology, and stated that the supposed phenomena arose from the touch of the operator calling into action muscles of expression, and thus arousing, by association, certain ideas in the mind of the subject. Sometimes the manifestations resulted from a previous knowledge of phrenology, or from training during hypnosis. In 1844 he showed that he was able to induce all the phenomena by verbal suggestion, no matter what part of the head was touched.

Suggestions, Passes, &c.—At first Braid employed mechanical methods alone, both for the induction of hypnosis and its phenomena. But, when he abandoned his physical theory, his views as to the value of passes, &c., naturally changed and he soon regarded them as only of secondary importance, and attached most value to suggestion. With certain impressionable subjects hypnosis might be caused by auto-suggestion; if they believed that something was taking place at a distance, capable of putting them to sleep, they would fall asleep, even if nothing were happening. Direct verbal suggestion, however, was best, both for the production of hypnotic phenomena and for the cure of disease. After hypnotising his patients, Braid stated in a confident manner the results he wished to obtain and, in certain subjects, found that these could be varied by simple change in the voice. Thus, if he made a patient see an imaginary sheep and then asked him, in a cheerful manner what colour it was, this tone usually elicited the reply "white," or some light colour. If he then asked, what colour is it now, giving a sad intonation to the word now, the reply would usually be "black."

The action of passes, &c., is explained by Braid in the following manner:—Everything which produces a new impression will modify or change existing functions, whether the new impression be of a mental or physical nature. The brain receives many impressions, which subsequently influence the mind, although they were not perceived when conveyed to it by the organs of sense and others, too slight ever

to become conscious, may nevertheless be sufficient to produce a local influence on the nerves and capillaries. Thus, a person may be engaged in reading, so as not to notice he is sitting in a draught, and yet this may cause rheumatism. Passes may have a mechanical action through the agitation of the air or by touch, or they may produce changes in temperature and electrical states. They are most powerful, however, when they directly excite mental action, either by fixing the attention on one part or function of the body and withdrawing it from others, or by arousing ideas previously associated with the physical impression. All these effects may be neutralised by direct suggestion, and it is quite possible, by a system of training, to make passes, &c., produce the opposite of the result usually attributed to them. Thus, "supposing that, with each touch or agitation of the air, the operator speaks aloud and predicts what should happen, the auricular suggestion may be so strong as to cause the predicted manifestation to be realised instead of what otherwise would have been the case; and thus, from this time forward through the double conscious memory, the like impression on that part or organ of sense will recall the previously associated idea of manifestation." Some sort of material combination may be sufficient to produce hypnosis when it is positively affirmed that it will do so, while a moment later the same method will produce nothing, when it is suggested it will be a failure. "All these phenomena may be realised without the patient intending to play off any deception on others, or having any remembrance of the fact on coming out of the sleep."

Dangers.—According to Braid, the belief that hypnotism might be used for immoral purposes had arisen through the mesmerists asserting that they could influence patients irresistibly, even at a distance, by mere volition and secret passes. He was certain no one could be involuntarily affected by hypnotism, in any stage of the process, and he could give instructions to even the most susceptible subject by means of which he could completely resist anyone. In his hypnotic experiments he had always observed caution and had neither met with the slightest accident, nor seen a

case which he could not awaken in two minutes. In passing into the hypnotic state, reason and will were the first mental powers to wane ; the cerebral spinal functions became more excitable as the controlling powers of the will were withdrawn, and thus the imagination gained the ascendancy. This interference with volition, however, had its limits, and in the alert stage, the patients were even more fastidious as regards propriety of conduct, than in the waking condition ; while from the lethargic state, they could be aroused at once to one of exalted sensibility, either by being touched, or by a breath of air ; nor was it necessary that this should be done by the person who had hypnotised them. Hypnotism, perhaps, would not make a vicious person virtuous, but certainly it would not make a virtuous one vicious, and there was more chance of extorting secrets by giving the patient a few glasses of wine than by hypnotising him. Braid contrasted the dangers of ether with those of hypnotism, and concluded in favour of the latter, for the following reasons :—All impropriety was strenuously resisted in hypnosis ; for example, a patient could not be induced to take off her stockings, or to give a kiss to a gentleman, even should he be a hallucinatory one ; on the contrary, she would repel such suggestions with more energy than in the waking condition. Hypnosis quickened the moral sense, while at the same time it rendered the patient docile and obliging in carrying out reasonable suggestions. Thus, while the patient would indignantly refuse to kiss an imaginary gentleman, she would be quite willing to do so to a child. Granting that it were possible to commit a crime during hypnosis, its detection would be inevitable, as the patient when again hypnotised would be certain to remember it ; but in ethereal narcotism, there would not be the safeguard of double consciousness, and many powerful medicines had been used for criminal purposes, without the knowledge of the victim. While Braid had never seen any excitement of the animal passions during hypnosis, he had frequently observed spontaneous and intense erotic manifestations arise during the primary stage of etherisation, even in the most modest and virtuous.

General Observations.—Braid desired to give full publicity to all his methods and results, and insisted that the phenomena should be examined in the most critical manner, with an honest desire to arrive at truth, as it was impossible to estimate them justly from mere reading or hearsay evidence. He had taken every care to avoid deception, but, as he had been sceptical himself, he did not expect his conclusions would be unhesitatingly accepted.

Braid did not regard hypnotism as a universal remedy, but believed it capable of curing many diseases in which ordinary treatment failed; it was also of use in order to obtain a correct diagnosis. In every case it could be applied with the greatest confidence and absolutely without fear of discomfort, pain or danger. But it was only medical men, well versed in anatomy, physiology and pathology, who were competent to use it with advantage and safety, and the ignorant were warned against tampering with such a powerful agency.

Despite the fact that Braid repudiated the possession of any mysterious force or power, he was more successful than the mesmerists in influencing his patients. He always endeavoured to strip hypnotism of mystery, and believed that he could teach any intelligent medical man how to employ it.

In reference to the almost incredible opposition, both of the orthodox practitioner and the mesmerist, he said: "Like the originator of all new views, however, hypnotism has subjected me to much contention; for the critics, from not perceiving the difference between my method and that of the mesmerists, and the limited extent of my pretensions, were equally hostile to hypnotism as they had been to mesmerism; and the mesmerists thinking their craft was in danger, that their mystical idol was threatened to be shorn of some of its glory by the advent of a new rival, buckled on their armour, and soon proved that the *odium mesmericum* was as inveterate as the *odium theologicum*."

Historical.—Braid died suddenly on March 25, 1860, according to some accounts from apoplexy, according to others from heart disease; he left a widow, son and

daughter. He maintained his active interest in hypnotism up to the last, and three days before his death he sent his last MS. to Dr. Azam, with the following inscription: "Presented to M. Azam, as a mark of esteem and regard, by James Braid, Surgeon, Manchester, March 22, 1860."

Sympathetic notices of Braid's death appeared in the local papers and different medical journals, all of which bore warm testimony to his professional skill and high personal character. The *Lancet* drew attention to the fact that, though he was best known in the medical world for his theory and practice of hypnotism, he had also obtained wonderfully successful results by operation in cases of club foot and other deformities, which brought him patients from every part of the kingdom. Up to 1841 he had operated on 262 cases of talipes, 700 cases of strabismus and 23 cases of spinal curvature.

The following are the titles of all the works and articles by Braid which I have been able to trace.

(1) "Satanic Agency and Mesmerism reviewed, in a Letter to the Rev. H. McNeile, A.M., in reply to a Sermon preached by him." (1842, 12mo.)

(2) "Neurypnology, or the Rationale of Nervous Sleep, considered in Relation to Animal Magnetism, illustrated by numerous cases of successful application in the relief and cure of disease." (1843, 12mo, pp. 281.)

(3) "The Power of the Mind over the Body; an Experimental Inquiry into the Nature and Cause of the Phenomena attributed by Baron Reichenbach and others to a 'New Imponderable.'" (1846.)

(4) "Observations on Trance or Human Hybernation." (1850.)

(5) "Electro-Biological Phenomena, considered physiologically and psychologically," from the *Monthly Journal of Medical Science*, for June, 1851, with Appendix.

(6) "Magic, Witchcraft, Animal Magnetism, Hypnotism, and Electro-Biology; being a digest of the latest views of the author on these subjects." Third edition, greatly enlarged, embracing Observations on J. C. Colquhoun's "History of Magnetism." (1852.)

(7) "Hypnotic Therapeutics, illustrated by Cases, with an Appendix on Table-Turning and Spirit-Rapping." Reprinted from the *Monthly Journal of Medical Science*, for July, 1853.

(8) "The Physiology of Fascination, and the Critics Criticised." (1855.) The second part is a reply to the attacks made in the *Zoist*.

(9) "Observations on the Nature and Treatment of Certain Forms of Paralysis." (1855.)

Articles in the *Medical Times* :—

(10) "Animal Magnetism," vol. v., 1841-42, p. 283.

(11) "Animal Magnetism," vol. v., p. 308.

(12) "Neuro-Hypnotism," vol. vi., 1842, p. 230.

(13) "Phreno-Mesmerism," vol. ix., 1843-44, p. 74.

(14) "Mr. Braid on Mesmerism," vol. ix., p. 203.

(15) "Observations on some Mesmeric Phenomena," vol. ix., p. 225.

(16) "Observations on Mesmeric and Hypnotic Phenomena," vol. x., 1844, pp. 31 and 47.

(17) "Case of Natural Somnambulism and Catalepsy, treated by Hypnotism; with remarks on the Phenomena presented during the Spontaneous Somnambulism, as well as that produced by various Artificial Processes," vol. xi., 1844-45, pp. 77, 95, and 134.

(18) "Experimental Inquiry whether Hypnotic and Mesmeric Manifestations can be adduced in Proof of Phrenology," vol. xi., p. 181.

(19) "Magic, Mesmerism, Hypnotism, &c., historically and physiologically considered," vol. xi., pp. 201, 224, 270, 296, 399, and 439.

(20) "Case of Natural Somnambulism, &c.," vol. xii., 1845, p. 117. (Article giving further history of case already reported.)

(21) "The Fakirs of India," vol. xii., p. 437.

(22) "Dr. Elliotson and Mr. Braid," vol. xiii., 1845-46, pp. 99, 120, and 141.

(23) "On the Power of the Mind over the Body; an Experimental Inquiry into the Nature and Cause of the Phenomena, attributed by Baron Reichenbach, and others to a 'New Imponderable,'" vol. xiv., 1846, pp. 214, 252, 273.

(24) "Facts and Observations as to the Relative Value of Mesmeric and Hypnotic Coma and Ethereal Narcotism, for the Mitigation or entire Prevention of Pain during Surgical Operations," vol. xv., 1846-47, p. 381, continued, vol. xvi., 1847, p. 10.

(25) "Observations on the Use of Ether for Preventing Pain during Surgical Operations, and the moral abuse it is capable of being converted to," vol. xvi., p. 130.

(26) "Mr. Braid and Dr. Elliotson," vol. xvii., 1847-48, p. 106.

(27) "Mr. Braid and Mr. Wakley," vol. xvii., p. 163.

(28) "Observations on Trance or Human Hybernation," vol. xxi., 1850, pp. 351, 401, and 416.

In the *Lancet* :—

(29) "Talipes," vol. i., 1841-42, p. 202.

(30) "Queries respecting the Alleged Voluntary Trance of Fakirs in India," vol. ii., 1845, p. 325.

In the *Monthly Journal of Medical Science* :—

(31) "Entire Absence of Vagina, with Rudimentary State of Uterus, and Remarkable Displacement of Rudimentary Ovaries and their Appendages, in a married Female, 74 years of age," vol. xvi. (third series, vol. vii.), 1853, p. 230.

(32) "Hypnotic Therapeutics, illustrated by Cases," vol. viii., third series, 1853, p. 14.

In *Edinburgh Medical and Surgical Journal* :—

(33) "Observations on Talipes, Strabismus, Stammering and Spinal Contortion, and the best Methods of removing them," vol. lvi., 1841, p. 338.

(34) "The Power of the Mind over the Body ; an Experimental Inquiry into the Nature and Cause of the Phenomena attributed by Baron Reichenbach and others to a 'New Imponderable,' " vol. lxvi., 1846, p. 286.

(35) "Facts and Observations as to the relative Value of Mesmeric and Hypnotic Coma, and Ethereal Narcotism, for the Mitigation or entire Prevention of Pain during Surgical Operations," vol. lxvii., 1847, p. 588.

(36) "On the Use and Abuse of Anæsthetic Agents, and the best Modes of Rousing Patients who have been too intensely affected by them," vol. lxx., 1848, p. 486.

In the *London Medical Gazette*, new series :—

(37) "Lateral Curvature of the Spine—Strabismus," vol. i., 1840-41, p. 445.

(38) "Stammering," vol. i., p. 445.

(39) "Cure of Stammering," vol. ii., 1840-41, p. 116.

(40) "On the Operation for Talipes," vol. ii., p. 186.

Braid is also stated to have contributed a "Case of Cæsarean Section" to one of the medical journals, but this I have been unable to trace.

In 1852, in the Preface to the Third Edition of "Magic, Witchcraft, &c.," Braid stated that he now gave his views in reference to all important hypnotic and mesmeric theories, and hoped, by this means, to make up in some measure for the delay in the publication of another edition of his work on "Hypnotism," which had long been out of print, and was frequently called for. "That call," he said, "I hope shortly to be able to respond to, with such fulness of detail as the importance of its subject merits; more particularly with regard to its practical application for the relief and cure of some forms of disease, of which numerous interesting examples will be adduced." At the conclusion of "The Physiology of Fascination, &c.," he also said: "It is my intention shortly to publish a volume, entitled, 'Psychophysiology: embracing Hynotism, Monoideism, and Mesmerism.' This work will comprise, in a connected and condensed form, the results of the whole of my researches in this department of science; and it will, moreover, be illustrated by cases in which hypnotism has been proved particularly efficacious in the relief and cure of disease, with special directions how to regulate the processes so as to adapt them to different cases and circumstances." Shortly before his death, Braid contemplated publishing a second edition of "Neurypnology" in France; this was never done, nor did the two proposed works above referred to, ever see the light. All Braid's books and articles are out of print; and of the former, Nos. 2, 3, 4, 6, and 7 (see list, p. 107-8), are alone to be found in the Library of the British Museum. I possess Nos. 1, 2, 4, 5, 6, and 8; also two long MS. letters addressed to "M. Highfield, Esq., Surgeon," dated re-

spectively October 28, and November 16, 1842. These letters contain an interesting *resumé* of Braid's views on the subjective nature of hypnotic phenomena; some account of the hyperæsthesia of the special senses; reference to successful cases, together with a denial of the alleged dangers of hypnotism, and of the supposed automatism of the subject.

In 1859, Dr. Azam, of Bordeaux, became acquainted with Braid's hypnotic work, and commenced to investigate the subject for himself; an account of his experiments, with much reference to Braid, appeared in the *Archives de Médecine* in 1860. About the same time Broca, who had obtained marvellous results with Braid's methods, read a paper on "Hypnotism" before the Académie des Sciences, which attracted much attention, and Velpeau presented a copy of "Neurypnology" to the same society; whereupon a commission, composed of members of the four sections of the institute, was formed to report on the subject. On hearing of this, Braid wrote to the Académie to say how much pleasure Azam's brilliant results and the action of the society had given him.

From this date, the subject of hypnotism was never lost sight of in France, but it was not until forty years after its original publication that "Neurypnology" was translated by Dr. Jules Simon, who stated that "Braid's researches produced for him numerous enemies, but despite this, he pursued them with the precision of genius, and was able to add artificial somnambulism to the pathology of the nervous system—a chapter which the investigations of the greater number of the modern neuro-pathologists have confirmed."

At the present day, hypnotism is rarely mentioned, either at home or abroad, without due credit being given to Braid for his re-establishment of the subjective explanation of its phenomena. But, apparently, little is known of his later works, and of the remarkable change which took place in his theories. For example, Dr. Bastian, in his article on "Braidism" in Quain's "Dictionary of Medicine," expressed his regret that Braid did not reject all the so-called phenomena of phreno-hypnotism. Professor

Romanes also appears to have been only acquainted with "Neurypnology," and mentions it alone in his article on "Hypnotism" in the *Nineteenth Century* for September, 1880. He refers to the fact that Heidenhain omits all reference to Braid, and maintains that it would be doing scant justice to "Neurypnology" to say that all Heidenhain's results had been anticipated. For, "in the vast number of careful experiments which it ['Neurypnology'] records—all undertaken and prosecuted in a manner strictly scientific—it carried the inquiry into various provinces which have not been entered by Heidenhain. No one can read Braid's work without being impressed by the care and candour with which, amid violent opposition from all quarters, his investigations were pursued; and how, when after the lapse of nearly forty years, his results are beginning to receive the confirmation which they deserve, the physiologists who yield it ought not to forget the credit that is due to the earliest, the most laborious, and the hitherto most extensive investigator of the phenomena of what he calls hypnotism."

The only English writer, as far as I know, who refers to Braid's later works is C. W. Sutton, in the "Dictionary of National Biography," where he states that some of them have been translated into French and German.

The following passage in "Suggestive Therapeutics," published in 1890, shows that, at that date at all events, Bernheim was unacquainted with Braid's advanced theories:—"Braid made use of suggestion without knowing it. We must come down to 1860 to find the doctrine of suggestion entirely freed from all the elements that falsified it, even in the hands of Braid himself, and applied in the simplest manner to therapeutics. The patient is put to sleep by means of suggestion. He is treated by means of suggestion. The subject being hypnotised, Liébeault's method consists in affirming, in a loud voice, the disappearance of his symptoms. Such is the method of therapeutic suggestion, of which M. Liébeault is the founder. He was the first to clearly establish that the cures of pain by the old magnetisers, and even by Braid's hypnotic operations, are not

the work either of a mysterious fluid or of physiological modifications due to special manipulations, but the work of suggestion alone."

The first translator of Braid was W. Preyer, Professor of Physiology at the University of Jena, who, in 1881, published "The Discovery of Hypnotism." This was a condensation of "Neurypnology," together with the translation of the pamphlet sent by Braid to Dr. Azam in 1860, and which had passed into the possession of Dr. Beard, of New York, who had lent it to Preyer. The same pamphlet is also translated into French, and forms an appendix to Dr. Jules Simon's "Neurypnology," published in 1883. It contains an interesting account of many of Braid's later views, and describes in plain and unmistakable language his use of verbal suggestion—a fact which renders almost impossible of comprehension the statements of Professor Bernheim on the subject.

In 1882, Preyer published "Hypnotism," which consisted of a translation of all Braid's works, with the exception of "Neurypnology" and "Satanic Agency."

In the *Zeitschrift für Hypnotismus* for December, 1892, Moll warmly acknowledged Germany's indebtedness to Preyer for his translations, and recognised that the later works were a distinct advance upon "Neurypnology," especially in their psychological explanation of hypnotic phenomena. At the same time, even he apparently failed to grasp how entirely Braid had forestalled the "Suggestion" theory of the Nancy School. He admits, however, that "notwithstanding that many researchers have referred to and valued Braid's writings, the modern works are only in the smallest degree a continuation of his."

The fact that Braid's later theories have been so entirely overlooked is all the more striking, when one realises the firm hold they had taken upon scientific opinion in his day. In illustration of this, it is only necessary to refer to the works of the late Drs. Carpenter and John Hughes Bennett. The former gives a full exposition of Braid's views in his "Principles of Mental Physiology," sixth edition, London, 1881, "Mesmerism, Spiritualism, &c.," and elsewhere. The

following passage from an article in the *Quarterly Review* for September, 1853, shows how thoroughly Dr. Carpenter understood the importance attached by Braid to suggestion : —“ The clue to the marvel [mesmerism] was soon found by Mr. Braid, in the concentrated operation of the principle of *suggestion* [the italics are Dr. Carpenter's] . . . and under the guidance of this idea, he has subsequently followed up the investigation with great intelligence, making no mystery of his proceedings, but courting investigation in every possible way.” Professor Bennett, in his “Text-book of Physiology,” reproduced the explanation of “Braidism,” both from the physiological and psychological side, first given by him in 1851. This formed a part of the lecture on hypnotism he gave annually to his students, in which he urged them to investigate a subject destined to revolutionise the theory and practice of medicine.

Above everything, it is worthy of note how Braid at once, by his earlier experiments, exploded the fallacies of the mesmerists, which, till then, had flourished successfully, despite the abusive and absolutely unscientific attacks of the combined medical press. In a later article, I hope to have the opportunity of showing that a thorough acquaintance with Braid's researches would have prevented many errors, both of the school of La Salpêtrière and that of Nancy.

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(1) “The New Theory on Animal Magnetism,” vol. v., 1841-42, p. 175. Editorial account of a lecture given by Mr. Duncan on Animal Magnetism, at the Hanover Square Rooms, on December 31, 1841. The lecturer, who had adopted Braid's views, explained them.

(2) “Animal Magnetism,” vol. v., 1841-42, p. 283. Editorial account of two lectures by Braid, one on March 1, 1842, at Hanover Square, and the other next day at the London Tavern.

(3) “On Mr. Braid's Experiments.” By Dr. Herbert Mayo, vol. vi., 1842, p. 11.

(4) “Mesmerism,” vol. vi., 1842, p. 47. Editorial account of two lectures delivered by Braid.

(5) “Hypnotism, or Mr. Braid's Mesmerism,” vol. x., 1844, p. 98. Letter to editor from “S.” in praise of Braid's researches and of the open-mindedness of the *Medical Times*.

(6) "Conversazione on Hypnotism," vol. x., 1844, p. 137. Editorial account of the Conversazione held by James Braid, at the Royal Manchester Institution, on April 22, 1844, by invitation of the Committee.

(7) "Jenny Lind and Hypnotism," vol. xvi., 1847, p. 602. An interesting account of Braid's experiments to show the power of somnambulists in imitating languages and song.

(8) "Operations under Hypnotism in Paris," vol. xix., 1859, p. 646. Account of Azam, with reference to Braid.

(9) "Mr. Braid, of Manchester," vol. i., 1860 (new series), p. 355. Obituary notice.

(10) "The late Mr. Braid," vol. i., 1860 (new series), p. 396. Letter to Editor from A. W. Close, F.R.C.S., exposing the absurdity of certain statements in the above account of Mr. Braid's death.

In the *Lancet* :—

(11) "Mr. Braid's new Operation for Club Foot," vol. i., 1841-42, p. 326.

(12) "Hypnotism in Paris," vol. ii., 1859, p. 650. Article referring to Azam, Velpeau, Broca, &c., and stating that they had copied the methods of Braid and Esdaile.

(13) "Sudden Death of Mr. James Braid, Surgeon, of Manchester," vol. i., 1860, p. 335.

In the *Edinburgh Medical and Surgical Journal* :—

(14) Critical article on Braid's "Observations on Trance, or Human Hybernation," vol. lxxiv., 1850, p. 421.

(15) "Abstract of a Lecture on Electro-Biology," delivered at the Royal Institution, Manchester, on March 26, 1851, by James Braid, vol. lxxvi., 1851, p. 239.

(16) "Death of Mr. James Braid, Surgeon, of Manchester," vol. v., 1859-60, p. 1068.

(17) "Case of Contracted Foot with severe pain, cured with Mesmerism," *The Zoist*, vol. iii., 1845-46, p. 339. Article by Elliotson, referring to Braid.

(18) "Researches in Magnetism, Electricity, Heat, Light, Crystallisation, and Chemical Attraction, in their relations to the Vital Force." By KARL, Baron von Reichenbach, Ph.D. Translated by William Gregory, M.D., London, 1850.

(19) Article V. *The British and Foreign Medico-Chirurgical Review*, vol. viii., July to October, 1851, p. 378. An article on Mesmerism, Magnetism and Hypnotism, with favourable reference to Braid and his views.

(20) "Electro-Biology and Mesmerism." *The Quarterly Review*, vol. xciii., 1853. Article VI., p. 501. This article, which is unsigned, is believed to be by Dr. Carpenter, and gives an interesting account of Braid's work and theories.

(21) "Death of Mr. Braid, Surgeon." *The Manchester Courier*, Saturday, March 31, 1860. This article gives an account of Braid's life and writings, and refers to his "Case of Cæsarean Section," which I have been unable to trace.

(22) "Monoideism." Text-book of Physiology, &c. By John Hughes Bennett, M.D., &c., 1871, part ii., pages 357 to 361.

(23) "Mesmerism, Spiritualism, &c." By William B. Carpenter, M.D., &c., 1877.

(24) "Principles of Mental Physiology, &c." By William B. Carpenter, M.D., &c. Sixth Edition, 1881. Both these works contain much reference to Braid's experiments and theories.

(25) "Hypnotism." By G. J. Romanes. *Nineteenth Century*, September, 1880, p. 474.

(26) "Braid, James." By C. W. Sutton. "Dictionary of National Biography," vol. vi., p. 198. London, 1886.

(27) "Hypnotism or Animal Magnetism." By Rudolph Heidenhain, M.D. Translated from the Fourth German Edition by L. C. Wooldridge, M.D., D.Sc. With a Preface by G. J. Romanes, M.A., F.R.S. Second Edition. London, 1888.

(28) "Suggestive Therapeutics. A treatise on the Nature and Uses of Hypnotism." By H. Bernheim, M.D. Translated from the Second and Revised Edition by Christian A. Herter, M.D. Second Edition. Edinburgh and London, 1890, pp. 205-207.

(29) "Braidism." By H. Charlton Bastian. "A Dictionary of Medicine," edited by Richard Quain, M.D., London, 1882, vol. i., p. 131. "Mesmerism." By H. Charlton Bastian, 1882, vol. ii., p. 972.

(30) *Journal du Magnétisme*. Rédigé par une Société de Magnétiseurs et de Médecins. Sous la direction de M. le Baron du Potet. Vol. iv., 1847, p. 209; vol. viii., 1849, p. 66; vol. x., 1851, p. 510; vol. xiv., 1855, p. 400; vol. xix., 1860, pp. 62, 105. Du Potet refers to Braid's methods and theories, and also gives an account of a somnambule in Paris who could imitate languages and song.

(31) "Note sur le sommeil nerveux ou hypnotisme." Par le Dr. Azam de Bordeaux. *Archives générales de médecine*, cinquième série, tome xv., p. 5. Paris, 1860.

(32) "Mémoires d'un Magnétiseur." Par Ch. Lafontaine. Tome Premier. Paris, 1860, pp. 311-314.

(33) "Neurypnologie. Traité du sommeil nerveux ou hypnotisme." Par James Braid. Traduit de l'anglais par le Dr. Jules Simon. Avec Préface de C. E. Brown-Séquard. Paris, 1883.

(34) "De la suggestion et du somnambulisme dans leurs rapports avec la jurisprudence et la médecine légale." Par Jules Liégeois, Professeur à la Faculté de Droit de Nancy. Paris, 1889. Liégeois gives an account of the theories of the Abbé Faria and Bertrand, pp. 17-23, and cites "De la cause du sommeil lucide ou étude sur la nature de l'homme," par l'Abbé Faria. Paris, 1819. "Traité du somnambulisme, et des différentes modifications qu'il présente," par M. le Dr. Bertrand, Paris, 1823, and "Du Magnétisme animale et des jugements qu'en ont porté les Sociétés savantes." Par A. Bertrand. Paris, 1826.

(35) "Mystères des sciences occultes." Par un Initié. Paris (no date). A short account of Braid is given, pp. 296-300, and at p. 289 is to be found the only portrait of Braid with which I am acquainted. (Facsimile d'une lithographie d'après nature, imprimée à Liverpool, en 1854.)

(36) "Die Entdeckung des Hypnotismus," von W. Preyer. Berlin, 1881.

(37) "Der Hypnotismus." Ausgewählte Schriften von J. Braid. Deutsch herausgegeben von W. Preyer. Berlin, 1882.

(38) "Literaturbericht," von Dr. med. Albert Moll in Berlin. *Zeitschrift für Hypnotismus*, December, 1892, p. 107.

J. MILNE BRAMWELL.

Reviews and Abstracts.

Anatomie des Centres Nerveux. Par J. DEJERINE, Professeur Agrégé à la Faculté de Médecine de Paris, avec la collaboration de Madame DEJERINE-KLUMPKE. Vol. I. Imp. 8vo, 816 pages, with 401 illustrations, 45 of which are coloured. Paris: Rueff et Cie., 106, Boulevard St. Germain, 1895.

THIS work is the most exhaustive consideration of the subject with which it deals, namely; the anatomy of the cerebrum, which has as yet appeared in any language. It is so complete, both in its facts and in its bibliography, that it ought to be in the hands of every teacher of anatomy and every neurologist.

We propose to limit this notice to an account of the contents of this volume.

The first chapter describes the methods generally employed in the study of the central nervous system. The mode of opening the spinal canal and the cranium, and the removal of the spinal cord and brain are first described, and then an account is given, and deserves careful consideration, of the various modes of section of the brain in the fresh condition which have been recommended by Virchow, Meynert, Pitres, Brissaud and others, and their various advantages and disadvantages are considered. The methods of hardening, embedding and cutting of microscopical sections are described in full detail, the authors using Gudden's microtome for the last purpose.

Fifteen pages are devoted to the various methods of staining, among which we regret to find no mention of Bevan Lewis's process, which, curiously, seems to be little known on the Continent.

The second chapter is concerned with the development of the central nervous axis, and is very fully illustrated with drawings, which are partly original and partly reproduced from His.

The third chapter discusses the development from the histological side, both of the central and peripheral nervous system,

while the fourth chapter discusses the general histology of the central nervous system. Both of these chapters are remarkably full, and include the most important results of the more recent methods.

The second portion of the volume contains the specially original portion of the work of the authors, namely, the anatomy of the cerebrum, and is most minute in its descriptions, and most beautifully illustrated with photographs and original drawings, which have been made in part from macroscopic sections, and in part from microscopic sections stained by the Weigert-Pal method; the accuracy and artistic finish of those drawings we have never seen equalled. A long chapter is devoted to the surface anatomy of the cerebrum, each convolution and its subdivisions, and each fissure being carefully described. The value of the account is greatly enhanced by giving the various synonyms in use by different authors, which are often very confusing. The interior of the cerebrum is illustrated, as already said, by macroscopic and microscopic drawings. Of the former there are sixteen horizontal, thirty vertical transverse, and ten sagittal sections. Of the microscopic sections there are twelve vertical transverse, and twenty-one horizontal, fifteen of which specially illustrate the structure and relations of the basal ganglia. Lastly, there are thirteen oblique longitudinal sections of the sub-thalamic region. The concluding chapters of the work are concerned with the minute structure of the grey and white matter of the cerebrum, the former of which have been studied specially by the method of Golgi.

We heartily congratulate the authors upon their completion of this monumental work, and trust that it may meet with the success that it merits.

ALEXANDER BRUCE, M.D.

Cahier de Feuilles d'Autopsies : Pour l'Étude des Lésions du Névraze. Imp. folio, 29 pages, with 25 illustrations.
Paris : Rueff et Cie., 106, Boulevard St. Germain, 1895.
Par Dr. DÉJÉRINE.

THIS book consists of a series of outline drawings of various aspects of the brain:—vertex, base, outer and inner aspects, horizontal sections at several levels of the cerebrum, vertical

transverse sections of the occipital lobe, Meynert's dissection exposing the upper surface of the corpora striata, the optic thalamus, and the cerebellum, as well as a series of horizontal sections through the corpora quadrigemina, the pons and the spinal cord. The drawings, which are made from nature, are intended to facilitate the description and localisation of lesions of the central nervous system during the course of a *post-mortem* examination, or during the subsequent examination of microscopic sections. A number of valuable hints are given with regard to the manner of making sections in the fresh state, some of which seem to deserve special attention.

Dr. Déjérine recommends the opening of the spinal canal and the removal of the cord before the opening of the cranium and the extraction of the brain. This method allows the cord to be divided transversely between the second and third cervical nerve roots, and thus avoids the loss of tissue produced by the oblique incision made from within the cranial cavity.

Fresh sections should not pass near any lesion, nor any part which is important for the study of secondary degeneration, because the surfaces of the fresh section swell up unequally under the influence of the hardening fluid, and thus a considerable proportion of tissue is lost. In cases of cortical lesion the brain should be hardened without removing the pia mater. The cerebellum should be removed by a transverse incision through the pons, passing below the posterior corpora quadrigemina, and above the point of emergence of the fifth nerve. The two hemispheres are then separated by an antero-posterior incision, which, in cases of unilateral lesion, should pass at least a centimetre to the sound side of the mesial plane. The hemisphere is then divided into three portions by two vertical, transverse incisions, the anterior of which passes through the knee of the corpus callosum, and just clears the tip of the temporal lobe; while the posterior incision passes through the hinder portion of the splenium of the corpus callosum. The middle portion is further divided into two by a longitudinal incision, which passes at the level of the groove between the caudate nucleus and the optic thalamus. If the lesion is central, it is better to make the original incisions in a longitudinal direction. The upper one should pass through the upper portion of the optic thalamus as just described, and, provided they do not intersect the lesion, additional sections may be made parallel to this at distances of about one centimetre from each other.

In lesions of the cerebellar cortex the basal ganglia of the

cerebrum, crura cerebri, and cerebellum may be hardened together, after being removed from the rest of the cerebrum by Meynert's method. On the other hand, if the cerebellar lesion is central, the lateral lobes of the cerebellum may be removed by two sagittal sections, passing through the flocculus and the middle cerebellar peduncle, external to the point of emergence of the fifth nerve. These directions appear to be of great practical value, especially if the brain be injected with Müller's fluid on three successive days from both carotids and one vertebral artery by Hamilton's method.

ALEXANDER BRUCE.

Atlas of the Human Brain, and Description of the Course of the Nerve Fibres. By Dr. E. FLATAU. With Preface by Prof. MENDEL. Translated by WM. NATHAN, M.D., and JOHN H. CARSLAW, M.D. Publisher: F. Bauermeister, Glasgow.

THE object of this work to provide the medical profession with an atlas of photographic illustrations of the normal human brain examined in the fresh condition, unaltered by preserving fluid, and true to nature in details and size, has been most admirably accomplished in the laboratory of Prof. Mendel by Dr. E. Flatau.

There are eight photogravure plates, beautifully executed by Meisenbach, Riffarth and Co., of Berlin, illustrating the various surfaces of the brain and sections through the organ. These include the base with blood vessels; the upper surface; the outer surface; and vertical, horizontal, and sagittal sections. Some of the plates contain two figures, and besides the above most artistic photographic reproductions there are thirteen clear valuable diagrams indicating the course of the afferent and efferent tracts, and the various projection and association systems of the central nervous system.

The illustrations have been chosen with care and discretion, and each figure is clearly numbered, while on the opposite page is a key to the numbers, and no difficulty will be found in distinguishing any of the structures referred to in the plates.

There are twenty-five pages of clear letterpress, translated by Drs. Nathan and Carslaw, which concisely set forth the course

of the fibres in the central nervous system, and embody a brief but clear account of—

(1) The projection fibres of the cerebrum and the cerebellum ; and

(2) The association fibres ; based upon the latest opinions, as formulated by anatomists and physiologists.

In his preface, Prof. Mendel “ hopes that the success of this atlas may be as conspicuous as the industry and care which has been bestowed upon it ” ; we cordially endorse this statement, and consider that it will be of great use to pathologists and neurologists who are engaged in examining diseased brains.

As it is seldom possible to have at hand for comparison a normal fresh brain in order to determine exactly the localisation of disease, this atlas will be especially valuable to those engaged in making *post-mortems*, particularly in asylums and hospitals for nervous diseases.

F. W. MOTT.

Les états intellectuels dans La Mélancholie. Par GEORGES DUMAS, M.D. Paris, 1895.

La Confusion Mental Primitive. Par PH. CHASLIN, M.D. Paris, 1895.

The Insanity of Over-exertion of the Brain. By J. BATTY TUKE, M.D. Edinburgh, 1894.

THESE three works on insanity, although treating of different forms of disease, are interesting from the fact that the writers approach their respective subjects from three widely differing points of view.

Dr. Georges Dumas' *brochure*, of 140 pages, is principally based on the clinical study of the association of ideas in the simpler forms of melancholia. After analysing cases, in some of which organic conditions, and in others intellectual causes were antecedent ; he traces the origin of the melancholic ideas in both to the endeavour of the sufferer to explain, by logical synthesis, the changed emotional state ; in other words, to the attempt made by the patient to co-ordinate the ego with the altered affective condition. Thus, if organic conditions are the cause of the

depression, reasons are sought for being depressed. If sad ideas possess the mind, these are justified in the melancholic state, by accessory ideas. So also in aboulia of action or choice; the inability to act or choose is justified or explained, and impulsive action, when beyond all reasonable explanation, is met by the assumption of another ego. The character of Hamlet is ingeniously analysed by the author, at great length, in illustration of these views.

The slowness and painfulness, with consequent limitation and monotony of the mental life, leading to weakness of perception and association, are described, as other modes of the invasion of the ego.

In treating of the relations of the intellectual and organic states in melancholia, he quotes the views of Sollier and Lange, on the connection between the peripheral sensibility and emotion. Sketching the numerous evidences of defective nutrition, and motor activity, &c., in melancholia, and comparing these with similar defects, induced, for example, by paludism (which is often accompanied by mental depression), he concludes that the cœnæsthesia resulting from the bodily state is the actual basis of the melancholy of organic origin.

The melancholy dependent upon ideas of a depressing character arises also, he maintains, by producing similar organic states, the confused perception of which results in emotional depression. The two forms, therefore, are based, he suggests, on the same organic condition; differing only in the order of their development. In support of this view, he quotes Meynert's theory, that physical pain is chiefly made up of motor and vascular reflexes; and argues that psychical pain also, when sudden, as in shock, is followed by mental confusion with movements, accompanied or succeeded by organic depression (fatigue, weakness, hyperthermia, &c.). Melancholia, therefore, whether from organic or intellectual antecedents, is based on the same organic conditions, and is always the consciousness of the state of the body as represented in the cœnæsthesia.

Dumas does not deal with the question why some griefs are not followed (as in his theory they should be) by melancholia, but by mania; nor with such cases as the one Greisinger describes—of a woman melancholic one moment, when her womb was displaced, sane when it was replaced; nor with the fact that the general bodily health may be long depressed, even beyond what is found in marked melancholia, without this result. Neither does he recognise sufficiently that, in paludism and con-

ditions of generally defective nutrition, the nutrition of the brain must be affected with the rest of the body. All these questions have to be answered before M. Dumas' contention can be accepted.

M. Chaslin devotes the first third of his work to the historical literature of mental confusion; three-fourths of this is allotted to French writers on the subject, and, in a brief *résumé*, he concludes that there are two forms—one primary and idiopathic, the other primary and symptomatic.

The idiopathic he describes as developing after a variable, usually short, period of premonitory bodily and mental disturbance, not differing from that preceding the other forms of mental disorder, except by the occurrence of brief attacks of confusion, of which the patient is conscious. The actual onset is sudden or slow, sometimes with exaltation resembling mania, or with agitated melancholia, or with various degrees of stupor, even resembling katatonia.

The appearance of the case is complicated by the combination or succession (with alternations more or less irregular) of the states of exaltation and depression. The essential basis of disorder, however, is mental confusion, with loss of orientation, and with or without delusions, hallucinations, or illusions.

The mean type of the disorder he regards as corresponding to what the Germans call “acute verwirrtheit.” The cases differ from melancholiacs in not offering a systematic negation, but in yielding a docile assent to all that they are requested or made to do.

The actions, the facial expression, the speech, the memory, and the perceptions, all show mental obscuration, so that the individual may even have no conception of time, place, or of his own name, age, &c.

Somatic troubles are also very marked, there being general weakness, exhaustion, and defective nutrition; the face pale or flushed, the pupils usually dilated or unequal, trembling of the tongue and muscles of the face, especially in speaking. The pulse small, feeble, and slow; often there is some rise of temperature even to 40° (C.); great emaciation, and the skin dry; the feet and hands often congested. The reflexes variable, movements weak, uncertain, and slow. The urine may contain albumen, sugar, &c., and the menses are arrested. In fact, grave defects of nutrition are always present.

The progress is variable, and the duration months or years; the result—recovery, chronic confusion, dementia, or death.

This description is necessarily, he says, slightly artificial, and so also of the other forms resulting from the different degrees of confusion, of nutritional defect, of excitement, or depression, or from the forms of delusion. If the confusion or intellectual arrest is very marked, the result is acute dementia, restless, or stuporous, if the somatic state predominates, forms result which simulate an infectious disease, and may be called typhoidal or meningitic. Between the two above extremes, a very acute form, of rapid onset, is "*le délire de collapsus*," in which there is a general excitement, extreme confusion, often intense fever, even assuming the aspect of acute mania. Beside this may be placed the "*confusions légères*" of Delasiauve, approximating to neurasthenia, and the more chronic form described by Meynert as *amentia*. Of these he gives a brief description.

The *primitive symptomatic mental confusion* may be associated with organic disease, or pathological variations of disorder; as examples he quotes cases arising from infectious diseases, especially typhoid, erysipelas, cerebral rheumatism, the acute delirium of infectious origin; the polyneuritic psychosis of Korsakoff; uræmia, diabetes and gout; heart disease, alcoholism, ergotism; meningitis, cerebral tumours, brain syphilis, and some cases of cerebral hæmorrhage; general paralysis, dementia ("precoce" and senile); as well as forms of cerebral softening; folie circulaire, epilepsy, and exophthalmic goitre; while some degree of it results from exposure to extreme cold (Larrey) and from starvation.

In dealing with the pathologic psychology of confusion, he refrains from discussing theories of association, since if synthesis of its elements be admitted as the basis of mental function its loss must be admitted as possible. Confusion, he asserts, is found to be the basis of the mental trouble, independent of hallucinations or delusions, and is constituted by the difficulty and perversion in the association of ideas, &c.

This defect of synthesis affects all degrees of association, from simple perception upwards.

Thus perception of objects is imperfect or perverted, and even where correct the higher reasoning cannot make use of it, or only imperfectly.

The intellectual operations are weakened; the association of ideas and verbal images is defective; imagination is weakened, and reasoning is difficult, or impossible. The memory is generally implicated, the voluntary reproduction of ideas being singularly troubled, as well as their conservation; the power of making new acquisitions does not exist; but automatic reproduction does

occur (even of facts occurring during the illness), but, then, as if unknown to the personality of the patient.

The will is also affected, the acts being difficult, incomplete and awkward; there is loss of the motor images; the synthesis necessary for a voluntary act is defective, and attention (one element of the synthesis) is weakened, words in speech and writing being ill-adapted, phrases and even words being unfinished.

Emotion (the synthesis of innumerable obscure, bodily sensations) is weakened by the disassociation, and is feebly felt; the physiognomy reflecting the mental and emotional uncertainty.

Automatism results from this mental disassociation, and finally abnormal syntheses complicate the condition resulting in the extreme in complete incoherence.

Mental confusion, he asserts, may not only appear primarily, playing the leading part in the disorder, and symptomatically, as before stated, but also secondarily on other psychical conditions.

This secondary confusion may result where the acceleration of ideation is so great that the ideas are transformed immediately into words and acts; or from the mere crowd and whirl of thoughts of which some patients complain; it may arise from numerous hallucinations (especially visual); from sudden emotional change (especially anger and anguish); and lastly, in great variety from intellectual feebleness, congenital or acquired.

He admits that many of these causes of confusion may occur in the same case, *e.g.*, in the "*délire de collapsus*," and that, consequently, both primary and secondary confusion may be found co-existent.

Primary idiopathic mental confusion, he concludes by defining as an affection, ordinarily acute, following on a cause, usually appreciable (in general an infection), which is characterised by somatic phenomena of denutrition, and by psychic phenomena primarily resulting from the somatic states.

The mental state being constituted by a form of intellectual enfeeblement and disassociation (confusion), which may be accompanied or not by delusions, hallucinations, &c., by agitation or motor inertia, with or without marked emotional variations.

In the chapter on diagnosis, he admits the impossibility of drawing a definite line of demarcation between this and the other forms of disorder; for example, he admits that there may be cases in which it is not possible to say whether the melancholia or the confusion are the predominant factors.

That a state of mental confusion dependent on defective synthesis is the primary and important feature in certain cases of insanity has been fully recognised, and that this defective synthesis while predominant in the cognitive aspect of consciousness, is also marked on the emotional and volitional sides, would also be generally admitted.

M. Chaslin demands the further recognition of this condition as the fundamental factor, in cases in which there are prominent emotional, delusional, or volitional characteristics, and in so doing makes a distinct advance in our views of mental disorder, from a symptomatological outlook, simplifying the arrangement of many cases hitherto ranked under various heads of the voluminous terminology of which a lengthy abstract is appended. Every such generalisation of symptomatology is a probable approach to pathological verity; since the actual abnormal conditions of brain are probably not numerous; the ultimate pathological categories, when arrived at, will certainly bear a minute numerical relation to the symptomatological forms of disorder which burden our textbooks.

If confusion be accepted as affecting the basal element (synthesis) of cognition, feeling and volition, it probably denotes a wider disorder of function than attains in states in which only one of these aspects of consciousness is specially involved; and this is confirmed and emphasised by the grave nutritional disturbance accompanying it.

M. Chaslin does not dwell on the prognosis; but as he regards it especially from the side of toxic causation, this, while unfavourable to life in the more acute forms, would be favourable to mental recovery, in proportion to the transient nature of the cause. Yet there can be little doubt that the cerebral condition is more grave than in less complex conditions of disorder.

The writer, in visiting an asylum some years since, was struck by what appeared to him to be the hopeless type of the cases of mania, owing to the presence, in their symptoms, of a great amount of mental confusion; this, however, on inquiry was found to be secondary to the action of a drug which was in vogue at that time, and the prognosis had to be modified in accordance with this fact. Similarly, those cases of melancholia and stupor which manifest much mental confusion are more hopeless than those in which the emotional or volitional arrest is the most striking factor.

The chapter on treatment calls for little comment, being merely a *résumé* of well-known views.

M. Chaslin is undoubtedly to be congratulated on having very carefully collated a very diffuse literature, in a clear and concise form, as well as on the fact of having made a useful generalisation of the confused and conflicting observations on this subject.

"The Insanity of Over-exertion of the Brain," by J. Batty Tuke, is a work of sixty-six pages only, being the Morison Lectures of 1894.

In these lectures Dr. Tuke has applied to his subject the results of recent advances in histological research in the cortical matter of the cerebrum, viz., those made by Ramón y Cajal, Golgi, Lewis, &c., together with the experiments of Hodge, on the effects of expansion on nerve cells.

The first lecture is a summary of the histological structure of the cortex, up to date, accompanied by very satisfactory coloured diagrammatic scheme of its membranes, blood vessels, lymphatics, nerve cells, fibres, &c., the whole being succinctly and clearly described.

In the second chapter, after alluding to Ray and Sherrington's observations, together with those of Langendorf and Geschiedlen, he concludes that the acid products of brain waste may so increase the irritability of the muscular wall of the vessels as to render it more susceptible to vaso-dilator or vaso-constrictor influence.

This lecture is also illustrated by photographs of sections by Hodge, showing very clearly the result of stimulation in the posterior spinal ganglion cell of a cat in four stages, and the morning and evening condition of the sub-œsophageal ganglion of the honey-bee.

He then sketches the course of origin of a subinflammatory condition, from irritation, through active hyperæmia, passive hyperæmia and congestion, together with the results from obstruction of the lymphatic system in accordance with the views of the latest histology. In support of this, specimens were exhibited, of which, however, no reproduction is given.

He then briefly summarises the symptoms in cases of insanity from over-exertion of brain function, and concludes that both mania and melancholia are dependent on common pathological conditions, from the fact that during the prodromal period excitement and depression alternate, may co-exist, may pass from one to the other state, even repeatedly as in folie circulaire; also from the similar variations in general paralysis and the sequence of psychological phenomena from poisons such as alcohol.

All this corresponds to the views long since expressed by Dr. Sankey, of the unity of the degenerative processes of insanity.

Dr. Tuke is probably quite right also in attributing the result, in mania or melancholia, in a given case to the intensity and rapidity of incidence of the irritation, in conjunction with the instability of tissue; and in admitting that inherent weakness of the ganglia of the vaso-motor system may have an influence in the rapidity of production of disorder: these also are views to which recent histology add greater probability.

In the third lecture he emphasises the predominant pathological changes in the Rolandic area, and hazards the opinion that the changes in the convolutions anterior to this region are indicative of impaired nutrition, rather than of active morbid action; "of interruption of trophesial function, which depends on the maintenance of the integrity of cell connection."

He also expresses the conviction that interruption of connection may be brought about by Wallerian degeneration of fibres, processes and collaterals, "the result of structural implication of cells, produced by over-stimulation, and maintained and aggravated by the effects of poisonous exudates."

The general diminution of several functional activities, accompanying such conditions, Dr. Tuke considers is probably due to impairment of the vaso-cerebral system, consequent on nerve implication; while admitting, however, that at present there is no definite proof of this, he quotes the analogy of certain forms of herpes and scleroderma.

He raises a protest against the possible errors in diagnosis and treatment, which may arise from a too facile employment of etiological classification, as exemplified by amenorrhœal insanity, &c.

The abuse of terms, however, it must be remarked, is not sufficient argument for their abandonment.

Dr. Tuke also asserts that for all practical purposes, "peripheral irritation may be dismissed from the list of producers of insanity," a view that does not savour of extensive observation of the early stages of the development of insanity. He also denies that there is sufficient evidence to warrant the connection of insanity with diseases of the heart, liver, or kidneys; although he accepts syphilis as a *vera causa*, he practically dismisses other diatheses.

The lecture on treatment calls for no special remark, except it be an expression of wonder that with the author's views, he is not more emphatic against the use of sedative and narcotic drugs.

In view of the fact that very competent observers have asserted that simple brain work is a rare cause of insanity, it

would have been satisfactory to have had some description of the causation in the cases which the author thus classifies.

In these three works we have mental disorder ascribed, by M. Dumas, to the peripheral cœnæsthesia, by M. Chaslin, to a toxic, hæmic condition, and by Dr. Batty Tuke, to functional overwork. That toxic conditions are sufficiently potent to produce such results there can be no doubt; in theory the first and last might be credited with the possibility of also producing such results. That the first one does do so is, however, extremely doubtful, and the last (overwork) is probably rarely efficient without other associated causes.

Careful studies such as these of the possible modes of action of individual pathological influences are to be highly recommended. They help us on the road to the comprehension of the share in causation of the complex causes almost invariably present in mental disorder, and of the equally complex pathological interactions in which they result.

SYNONYMS OF MENTAL CONFUSION FROM CHASLIN.

- Démence aiguë: Esquirol, Brierrè de Boismont.
 Stupidité, stupeur: Georget, Ferrus, Delasiauve, Dagonet.
 Confusion, confusion hallucinatoire: Delasiauve.
 Délire de dépression: Lasègue (?)
 Délire d'inanition: Becquet.
 Torpeur cérébrale: Ball.
 Acute primäre verrücktheit: Westphal.
 Hallucinatorischer Wahnsinn: Krafft-Ebing.
 Einfache Verwirrtheit: Wille.
 Acutes asthenische Delirium: Mayser.
 Acuter Wahnsinn: Schüle.
 Hallucinatorische Verworrenheit: Konrad, Salgo, Scholz.
 Asthenische u. hallucin. Verwirrtheit: Kraepelin.
 Hallucinatorisches Irresein: Fürstner.
 Dementia generalis acuta or subacuta: Tilling.
 Mania hallucinatoria: Mendel.
 Amentia: Meynert, Serbsky.
 Dysnoia, polyneuritic psychose: Korsekoff.
 Délire sensoriel: Schereschanski.
 Folie générale: Rosenbach.
 Paranoïa acuta or hallucinatoria acuta: various authors.
 Primary confusional insanity: Spitzka.
 Acute hallucinatory confusion: Spitzka.

Stupor, delusional stupor : Hayes, Newington.
 Acute confusional insanity : Conolly, Norman.
 Frenosi sensoria acuta, confusione mentale, amenza : Morselli.
 Stupidità : Morselli.
 Delirio sensoriale, Del Greco, de Luzenberger.
 Confusion mentale primitive : Chaslin, Séglas, &c.
 Paranoïa dissociativa : Th. Ziehen.
 Delirium hallucinatorium : Mendel.
 Collapsodelirium : Kraepelin, Aschaffenburg (H. Weber).
 Erschöpfungsstupor : Schaefer (?).
 Acute heilbare Dementia : Binzwanger.
 Dementia acuta : several authors.

Traité de Chirurgie Cérébrale. Par A. BROCA et P. MAURIAC. With 72 figures in the text. Masson et Cie., Paris, 1896.

MM. BROCA and Mauriac have written a treatise on cerebral surgery, based upon thirty-one cases which occurred in the hospital practice of M. Broca. The book contains a very complete bibliography, and published facts in cerebral surgery are cited with exactness. The work commences with the anatomy and physiology of the brain, and especially of the convolutions. Considerable space is devoted to the terminology and to the surgical guides for operative measures. From the commencement the authors show a very extensive clinical knowledge; and they devote themselves especially to pointing out in a very clear manner the indications and directions for operation rather than to technical details.

We may also note that the anatomical description is enriched by a number of very valuable, as yet unpublished, descriptions of the convolutions of the brain by the illustrious P. Broca.

In the second part, we find successively the study of traumatic lesions, complications of otitis, tumours, atrophic encephalopathy of infants, hydrocephaly, microcephaly, epilepsy, and encephalocoele. Concerning hydrocephaly and microcephaly the authors are especially in favour of operative interference, and M. Broca reports a successful case of drainage of the ventricles for hydrocephaly. Especially valuable is the chapter which deals with the

complications of otitis, and M. Broca, from an extensive experience demonstrates that with practice the most favourable method of dealing with all intracranial complications of otitis is by first opening up the mastoid cells. This book is well got up, and contains seventy-two figures. It should be read by all interested in cerebral surgery.

Atlas der Pathologischen Histologie des Nervensystems. V. LIEFERUNG. Lésions des cordons postérieurs d'origine exogène. Mit 10 Tafeln in Heliogravure par G. MARINESCO. Berlin, 1896. A. Hirschwald.

THE author, after considering the origin of the neurons, tries to give a natural classification of the diseases of the spinal cord. He divides the affections of the spinal cord into—

- (1) Lesions of the sensory neurons, direct.
- (2) Lesions of the sensory neurons, indirect.
- (3) Lesions of the motor neurons, direct.
- (4) Lesions of the motor neurons, indirect.

These lesions exist either pure or associated. All lesions of the neurons end in sclerosis.

If the lesion commences in *the neuron*, we have "parenchymatous degeneration," terminating in sclerosis. If, however, the lesion is primarily in the *vessels*, we have vascular sclerosis.

Finally, if the degeneration commences in the *interstitial substance*, we have neuroglia sclerosis, *e.g.*, syringomyelia.

The type of direct parenchymatous degeneration of the sensory neuron terminating in sclerosis is tabes.

The authors of this theory, in opposition to the vascular or interstitial theories, believe that parenchymatous degeneration ensues owing to the action of a toxin (syphilitic) upon the ganglion cells of the posterior spinal roots or the collaterals in the spinal cord.

Then follows an explanation of the plates, which represent tabes, cervical tabes, asymmetrical tabes, atrophy of Charcot-Marie, experimental lesion of a single root in animals, and accidental in man.

The author indicates with care the lesions of the collaterals—a hitherto neglected fact. The preparations were made in the laboratory of M. Charcot, at the Salpêtrière.

We can congratulate the author upon this work, which has been executed with great care and exactness. The preparations are faultless, and thus the very beautiful photographic reproductions clearly show all the interesting facts the author so lucidly yet briefly sets forth in the explanatory text. This work is of importance also for the classification the author gives of diseases of the spinal cord, based upon modern views of the structure of the nervous system, and also for those original observations with regard to lesions of collaterals.

This work cannot but be appreciated by all neurologists, and we can cordially recommend it to the readers of *BRAIN*.

Dana on the Localisation of Cutaneous and Muscular Sensations and Memories. (A Study of the Functions of the Motor Area of the Cortex of the Brain.) *Journal of Nervous and Mental Disease*, December, 1894.

In an article on "The Cortical Representation of the Cutaneous Sensations" (*Journal of Nervous and Mental Disease*, October, 1888), Dana stated that the clinical and pathological evidence so far collected showed that the motor areas of the cortex contain also the representation of cutaneous sensations; and that the sensory centres for the different parts of the body appear to be larger and more diffuse than the motor. After reviewing the experimental, the clinical and pathological, the anatomical and embryological evidence, and the evidence from teratology, he said:—"I am certain that no amount of scrutiny can explain away the numerous cases in which superficial cortical lesions have caused monoplegias and monoanæsthesias." He believes that the experience since accumulated has confirmed this position. Bastian, in his work on "Paralysis, Hysterical and Functional," has well said, however, that while the sensory function of the so-called motor cortex is acknowledged, the exact nature of this sensory function remains yet to be determined. We have yet to learn the part taken by these areas of the brain in tactile, thermal, muscular, and pain sensations. Dana again goes over this subject, and tries to determine more exactly the sensory functions of this much disputed region. In an article contributed to this subject by Hale White (*Lancet*, September, 1893), the author states that the conditions necessary to furnish perfectly good evidence pathologically of the function of the

central convolutions are very exacting: the lesion must be somewhat limited, it must be definitely localised, its nature must be known by a careful autopsical record, and the clinical phenomena caused by it must have been carefully studied. Very few recorded cases answer all the requirements; still, there are a few that were observed before the first article was written, and there have been some since. There have also been a number of cases in which a certain amount of positive and reliable evidence has been furnished, and careful experimental observations have been made.

Experimental Evidence.—Those who wish to study this point are referred to the very complete and thorough article by Mott (*British Medical Journal*, September 23, 1893, p. 685). Mott's experiments were made upon monkeys. He excised certain parts of the motor area; or, rather, he simply cut the cortex from the white matter by introducing a brain-knife bent at an angle. He afterwards tested the amount of paralysis and the defect in sensibility by the clip test, and he found that defective sensibility invariably occurred on the side opposite the lesion.

If the whole leg area were removed, there was paralysis of the opposite leg, permanent as regards the fine movements of the foot, diminished sensibility of the limbs to all forms of stimulus for some days after the operation, and permanent blunting of sensibility to the pressure of a weak clip fixed on the sole of the foot. This was tested on three animals. When the whole motor area was separated, there was permanent paralysis of the hand and foot, and paresis of the other muscles. Such animals showed, for some time after the operation, defective sensibility; only very painful pricking or heat would cause a response, and the parts permanently paralysed were also permanently anæsthetic. Mott infers from his experiments that a permanent defect of tactile sensibility, especially in the hands and soles of the feet, occurs after large lesions of the motor area, something akin to the paræsthesia of ataxia. He found in these cases degeneration of the internal capsule and the striæ medullares of the optic thalamus, with some degeneration of the corpus callosum. There was no degeneration in the posterior longitudinal bundle or the lemniscus, and there was no injury of the gyrus fornicatus.

Schæfer also found that removal of a part of the anterior central convolution produced defect in sensibility as well as motion. He also observed that the area removed must be of a certain size in order to produce this sensibility disturbance; in other words, that the excision of a very small motor area might

produce some motor defect without any detectible sensory defect. Horsley's experiments led to similar conclusions (*Nineteenth Century*, June, 1891).

Clinical and Pathological Evidence.

*Case 1 (personal).—*Male, aged 46. History of syphilis ten years before. Patient developed headache, convulsions of the left side of the body, followed by general convulsions and coma. Recovery from the convulsions, with a residuum of complete left hemiplegia and complete hemianæsthesia. Death.

Autopsy showed a syphilitic gumma of the dura mater pressing into and involving the central convolutions. The gumma was located about the middle portion of the fissure of Rolando, and was about one and a half inches in diameter.

*Case 2 (personal).—*Male, aged 55. History of syphilis. Gradual development of right hemiplegia, the paralysis being considerable in extent, and associated with some loss of tactile and pain sensibility on the right side. Death.

Autopsy.—Tumour size of hen's egg in middle of left ascending frontal convolution.

Case 3 is one of exceptional interest. *Sarcoma, involving hand and arm centre, paralysis, sensory symptoms, Jacksonian epilepsy. Operation. Great improvement.*

Boy, aged 16. At the age of thirteen a blow was received on the head, followed in six months by general epileptic convulsions. Later these became limited to left side, with aura beginning in fingers. A year before operation left arm became gradually weaker, with pains and twitchings in it; intense headache; vomiting and optic neuritis developed. Examination, May 29, 1894, showed paresis of left arm, with very slight paresis of left leg and face; exaggerated reflexes on left side. Sensory condition: Left hand and arm showed diminished sense of contact and pressure; sensory circles enlarged to 2 ctm. (he felt two points as one in this area); localisation of point on hand or arm touched defective; also, defective sense of position; cannot co-ordinate well (partly from paralysis); fumbling and clumsiness; weight sense defective; does not recognise nature of objects placed in hand; temperature sense normal; pain sense slightly exaggerated; no sensory troubles in the leg or face.

Operation by Dr. Conway: Trepanning over middle of anterior central convolution; removal of bone over an area three inches by two and one-half (see fig. 1b). Beneath the dura was found a flat

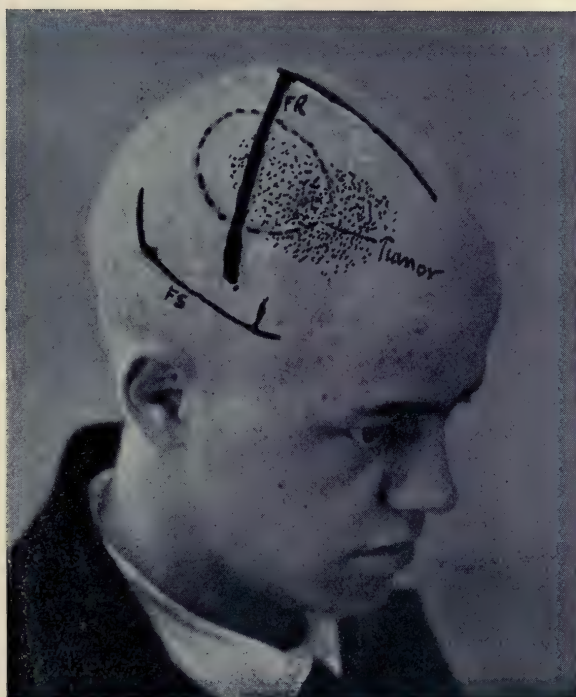


FIG. 1A.

Case 3. The situation of the trephine opening and of the tumour (from a photograph taken a year after the operation). The dotted line indicates the outline of the trephined area ; the dotted area indicates the position of the tumour.

Four months later: Paralysis still less; sensory condition about same; no pain; no headaches; *optic nerve normal*; no convulsions. General condition excellent. Patient eloped.

One month later, November 3, 1894, the boy returned, having had several epileptic attacks. He still has no headaches, or eye symptoms. The paralysis and sensory disturbances are about the same.

Case 4.—Amelia B., aged 17, single. When six years old had a fall and was struck on the left side of the head. This was followed a week later by a right-sided convulsion. Has since then had convulsions of an epileptic character with loss of consciousness, but always confined to the right side. They increased in frequency to one or two weekly. With them mental deterioration set in, and she became nearly imbecile. Her attacks were unilateral, and began in the right arm, which was raised over the head. Then the head and face and leg were involved. Lately the attacks became general.

Status presens.—There was slight weakness on right side, in arm and leg; no anæsthesia or ataxia. The knee-jerks were feeble on both sides, and so were the elbow-jerks.

She was operated upon on September 26, 1894. A piece of bone was removed two inches by one and one-half from over the middle third of the anterior central convolutions, the area extending a little above the middle third and anteriorly over the base of the second frontal convolution (fig. 2).

On removing the bone and opening the dura, both of which were normal, the brain tissue in the anterior part of the wound was seen to be dark and disintegrated. The extent of this could not be exactly ascertained without cutting into the brain unnecessarily. Its superficial area was about one inch in diameter, but might easily have extended down and forwards considerably. A strip one inch long and one-fourth inch wide was removed, and the wound sewed up.

As this case is not reported for its general pathological and clinical interest, further details are not given now.

Three days after operation she had had three right epileptic attacks. Examination at this time showed slight hemiplegia, most marked in lower half right face, less in right arm, least in leg.

Sensory.—Tactile anæsthesia was present in the right forefinger, where she could not feel a touch. It was slightly present on the rest of the hand, less still on the face; no anæsthesia of the leg. Analgesia was present in same area as the tactile anæ-

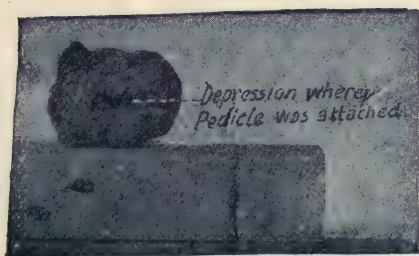
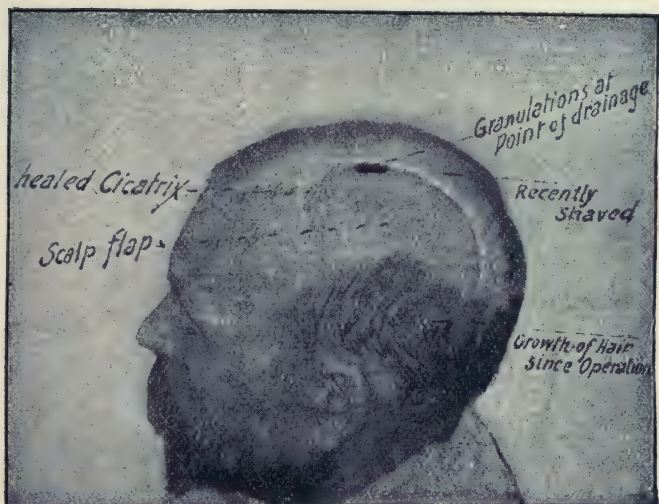


FIG. 3.

Case 5. Showing location and size of tumour in Dr. Steele's case.

thesia. There was no heat or cold anæsthesia. There was great clumsiness and awkwardness in moving the fingers and arm, and a slight difficulty in localising touch. She knew the position of the limbs and fingers.

She could not tell the nature of familiar objects placed in the hand. She could carry the finger to the point of the nose quite accurately.

The striking symptoms were the clumsiness and inability to handle and recognise objects, and the tactile and pain anæsthesia.

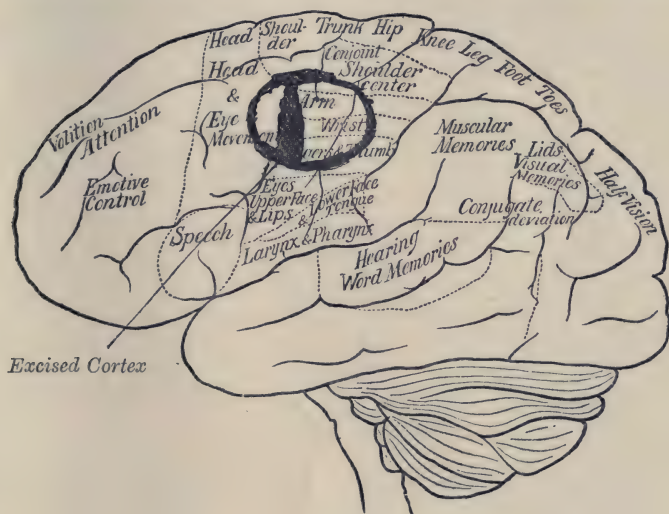


FIG. 2.

Case 4. Showing the area of bone removed and the position of the diseased cortex excised.

Three weeks later slight paralysis remained, the sensory symptoms had disappeared, except a slight awkwardness (more than could be explained by the paralysis). She had had no more convulsions, she seemed brighter mentally and was feeling well.

Case 5.—Male, aged 41. Gradual development of pain and paræsthesia in right leg, with Jacksonian epilepsy. At the end of a year the whole right side was affected. Examination then showed right hemiplegia and “sensory blunting” of right side. General symptoms of brain tumour. Operation: A fibro-sarcoma two and one-half inches by two inches was “shelled out” from the region of the upper third of the Rolandic convolutions (fig. 3). The tumour was attached to the falx by a pedicle, and

"grew from the wall of the longitudinal sinus." From this point of origin it grew into the Rolandic fissure, displacing the paracentral lobe. After the operation "sensation was first felt in the shoulder muscles, the next day he could move the arm, and on the third day he could draw up the forearm; and now he has good control of the whole arm except the fingers. He has no power, and but little sensation, in the fingers." He was discharged improved, and improving.—Steele, *Journ. of Amer. Med. Assoc.*, January 27, 1894.

Case 6.—This case is reported under the title of a tumour of the cortex, producing hemiplegia, with loss of tactile, pain and muscular sense. A man, aged 51, began to have local epilepsy in October, 1890, the convulsion beginning with paræsthesia, and paralysis marked in left arm. He began to lose tactile, pain and muscular sense in left arm and hand. Loss of tactile sense was absolute in the ring finger. The arm grew weak and the leg began to drag. Gradual increase of paralysis and general symptoms of brain tumour. Death in six months. Autopsy showed a softened melano-sarcoma 1 inch by $1\frac{3}{4}$ inches. "The tumour involved the posterior central, superior parietal, supra-Sylvian anterior portion of angular and occipital convolutions of the right side." The special point is the simultaneous involvement of the arm and hand in paralysis and anæsthesia.—Madden, *Journ. Nerv. and Ment. Dis.*, February, 1893, p. 125.

Case 7.—Man, crush of skull just behind right parieto-frontal section, followed by delirium and left hemiplegia. Exploration showed fracture of base as well as vertex. Removal of bone and contused brain over an area four by three inches. No rise of temperature. Return of consciousness on sixth day. Temporary left facial hemiplegia. Paralysis of left arm and leg improved up to a point, so that he could walk well, and left arm showed "good average power." "Voluntary movements were performed with left limbs in extremely awkward manner. He had complete loss of sense of position in the leg, and incomplete in the arm." "He could move the muscles of his face, but could not make a specified movement." Eye muscles normal. "Sense of resistance almost absent." Sense of weight not tested. On the affected side he had sensation of very rudimentary description, these being only tactile (?) and painful sensibility. He could not locate a touch or feel what touched him, but he knew there was contact.

He felt a pinch as painful, but could not locate it. Delayed sense of contact on face, less on arm. He could not distinguish

heat from cold, but if the heat was great it caused a diffuse sense of pain. In the leg the symptoms were similar, but less marked. Other senses normal.

The following parts of the cerebrum would appear to be involved: Lower half of ascending frontal, greater part of sigmoid flexure, posterior third of lower and middle frontal and base of upper frontal. Also, the base of the corresponding part of the falciform lobe. The motor centre for the arm was principally involved (fig. 4).

Electrical tests were made with the galvanic current, one pole on the neck, the other, a small olive-shaped electrode, was placed

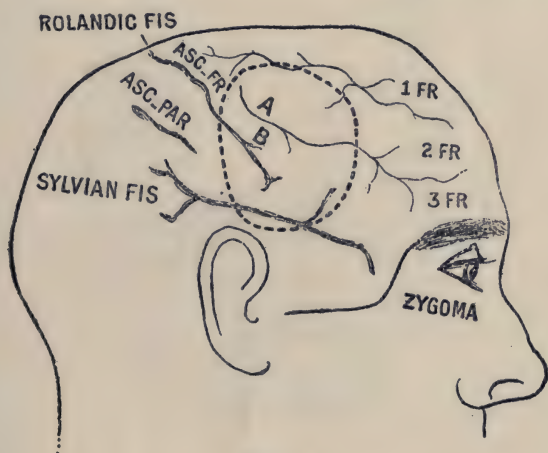


FIG. 4.

Case 7. Laycock. The dotted line shows the extent of the injury to the bone, the letters A and B are placed over the deepest part.

over the face, arm and leg centres. With currents of 5 to 10 ma., paræsthetic sensations were felt over the corresponding segments of the affected side. With stronger currents, 12 to 13 ma., both sides were affected, the right less than the left. There was no pain and no motor reaction. The negative pole gave far more definite results.

Later sensations of burning pain, cramp, and weakness were developed.—Laycock, *BRAIN*, 1893, vol. xvi., p. 605.

Case 8.—C. M., aged 68; tumour of the brain. Focal symptoms were weakness of the left leg, great weakness of the left arm and at the left angle of the mouth; exaggerated knee-jerks.

Sensory Symptoms.—There were slightly diminished tactile,

markedly diminished and altered painful, and much diminished thermal sensations all over the left arm and leg and left part of the face and trunk; the sense of position being also very imperfect in the arm and leg, especially the former. No hemiopia and no other affection of any of the special senses. Gradual increase of symptoms, and death about four weeks later.

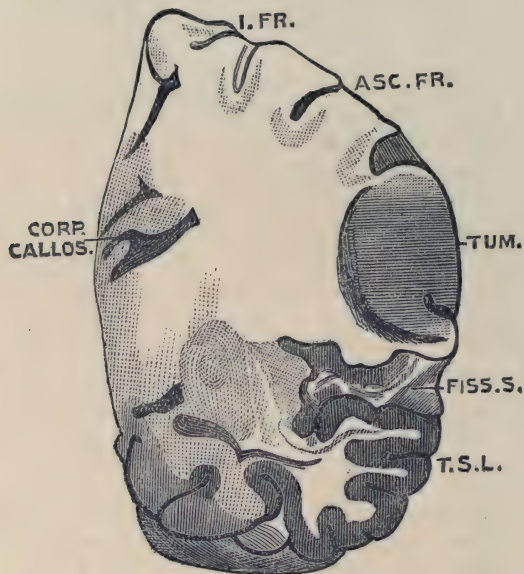


FIG. 5.

Case 8. Scirrhus of brain.

Tracing from a photograph, actual size, of the vertical transverse section passing through the lower end of the right fissure of Rolando. TUM., Tumour. FISS. S., Posterior limb of the fissure of Sylvius. T. S. L., Temporo-sphenoidal lobe. I FR., Superior or first frontal convolution. ASC. FR., Ascending frontal convolution. In the temporo-sphenoidal convolutions the grey cortex comes out well, but in the upper part of the section the details are very indistinct, owing to the white softening affecting this part.

Post-mortem.—A tumour was found in the right hemisphere, the size of a walnut and hard in consistence, surrounded by a margin of softened brain tissue. It involved the cortex for about an inch and was situated about an inch above the lower end of the fissure of Rolando, entirely replacing the grey matter. The growth was limited to the ascending frontal and parietal convolutions, both of which it invaded at their lower ends (fig. 5). There was white softening of the whole centrum ovale, which it may be assumed was of a date later than that when the examina-

tion that I have described occurred. The tumour was a scirrhus carcinoma. The history of the case showed that the disease began in the face centre. It was accompanied with epileptiform attacks which were associated with a sensory aura.—Wilson, *Lancet*, February 27, 1892, p. 463: "In passing it may be remarked that the occurrence first of a sensory aura (pins and needles) immediately followed by clonic spasm in the same parts, is of interest in connection with the theory that the motor area of the cortex contains sensory cells."

Case 9.—Male, 39. For two years Jacksonian epilepsy involving right leg chiefly, but with pain and spasm in right hand. Later, "constant and excruciating pain" in right hand and fingers, slight weakness of right side, right arm in rigid flexion, "tactile sensation fairly acute," skull opened over middle of left central gyrus and posterior part of third frontal. Small tumour lay on cortex. Cortex decorticated one-fourth inch deep over area larger than a dollar in the anterior two-thirds of opening. Next day right hemiplegia most in hand, no more pain in hand. "Sensation fairly acute." Can't speak. On seventh day, less paralysis. "With closed eyes cannot locate a pin prick on right side, but feels it promptly and keenly." Later, relapse. Some return of pain and convulsions. Paralysis improved.—Frank and Church, *Amer. Journ. Med. Sciences*, July, 1890.

Case 10.—A patient with Jacksonian epilepsy and symptoms indicating cortical tumour. Operation; no tumour found. Resection of cortex for space $1\frac{1}{2}$ by 1 cm. No fever. Patient developed a complete hemiplegia, which disappeared at the end of a month, "leaving only a slight anæsthesia and ataxia of the fingers, which prevented him from pursuing his occupation as a tailor."—Dunin, *Verhandl. des X. Intern. Med. Congress*, Band iv., p. 11.

Case 11.—Male; cortical degeneration, result of an injury. Before operation, sensibility normal. During operation of trephining cortex of left ascending parietal on level with first frontal gyrus was somewhat lacerated. A small bit of cortex, 3 by 6 mm. and 2 mm. thick, was excised. Entire injured surface had a diameter of not over $1\frac{1}{2}$ cm. For at least seven weeks after operation patient had "anæsthesia of right arm up to elbow, for touch, pressure, motion and position, sensibility to pain, heat and cold was retained." There was also impairment of highly purposive movements, inco-ordination and a little athetosis.—P. C. Knapp, "Intra-Cranial Growths," p. 61.

Case 12.—Man, aged 44. Received a blow on the left side of the head, causing a comminuted fracture. It was followed by a paralysis of the right shoulder and arm, with pain. Operation with removal of bone and contused brain tissue (fig. 6).

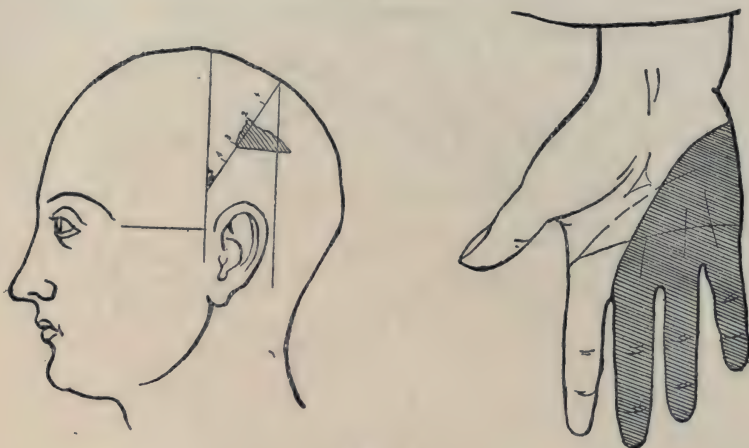


FIG. 6.

Case 12. Dr. J. Lynn Thomas. Showing location of scar with reference to fissure of Rolando, and the area affected with local hyperidrosis.

The wound healed and paralysis gradually improved. Later it progressed again, and six months after the wound the situation was as follows:—Weakness of left arm and hand; paralysis of the interossei and abductor of the little finger. Diminution of tactile sensibility in the upper third of the forearm. Two years later he could flex the fingers and use the hand to write. Tactile sensibility was lessened in the forearm and hand; sensibility to pain lessened in same region. Thermic sensibility slightly increased. Some hyperidrosis of the right hand.—J. Lynn Thomas, *Brit. Med. Journ.*, February 24, 1894, p. 400.

Case 13.—Male, aged 34, gradual development of right brachial monoplegia with symptoms of brain tumour. The lower extremity and face were not at all involved. Patient had phthisis. There was “a very marked diminution of sensibility of all forms, in the periphery more than in the parts near the trunk.” No disturbance of sensibility, except in the arm.

Post-mortem showed a tubercular tumour in the white matter just below the arm area of the cerebral convolutions. Its exact

position is shown in the accompanying figures, taken from the original (figs. 7, 8).—L. Darkschewitz, *Neur. Centralbl.*, vol. ix., 1890, No. 23.



FIGS. 7, 8.

Case 13. Showing location of tubercular tumour in Darkschewitz's case.

Case 14.—Male, aged 40. Traumatic hæmorrhage of pia mater, compression of Broca's convolution and of middle central convolutions, aphasia, partial right hemiplegia and hemianæ-

thesia. Trephining. Clot found and removed. Arm totally paralysed; tactile and pain senses impaired on paralysed side. Three years after operation paralysis of right hand remains, "with marked diminution of tactile and pain sense. . . . The muscular and temperature senses are perfect" (fig. 9).—Starr, "Brain Surgery," Case 18, p. 161.

Case 15.—Boy, aged 12. Right hemiplegia with athetosis, due to fall at age of 2. Trephining and cavity in middle of anterior cerebral convolution incised and drained. After operation, hemiplegia the same; "the right hand as high as the

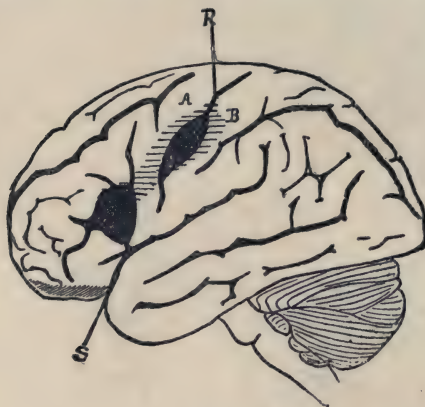


FIG. 9.

Case 14. Starr. Diagram of the left hemisphere of the brain, showing the situation of the clot.

wrist was decidedly anæsthetic to touch, temperature and pain, but there was no affection of the muscular sense" (fig. 10). One week later anæsthesia much less, athetosis gone.—M. A. Starr, "Brain Surgery," p. 38, Case 5.

Case 16.—Boy, aged 15. Blow on head, followed in twenty-four hours by paresis of right arm and leg, but not of face or arms. "The grasp of the right hand was less than half that of the left. The pain sense, while not destroyed, was greatly inhibited. The temperature sense was somewhat interfered with. His arm could be moved, but none of the delicate movements of fingers, hand, or forearm could be made. He could not tell the difference by touch between my silk handkerchief and the woollen blanket on his bed. A half-dollar put in the palm of his hand he called a dollar. He said a wooden dumb-

bell, weighing half a pound, weighed about five pounds. The right foot, toes, and leg were somewhat more paralysed than the arm. The sensation was in nearly the same condition as in the arm."

Dyslexia, agraphia, intelligence good. He could only walk with help. Gradual intensification of symptoms with headache



FIG. 10.

Case 15. Starr. The situation of the opening made in the skull.

until fourth day, when operation was done. Depressed circular fracture formed as indicated in inner circle (fig. 11).

A tarry, firm, extradural blood clot three-quarters of an inch thick at centre of circle found and removed. Gradual improvement—the muscular sense being last to recover. He was well three months after operation.—Hudson, *Annals of Surgery*, 1893, p. 421.

Case 17.—Male, 25. Blow on head, followed by right hemiplegia and complete motor aphasia, with twitchings of muscles of right hand and arm. He experienced some sensation in arm and leg, but apparently none in the face. Operation second day, removal of intradural clot, three ounces of blood removed; clot was three inches square and a quarter of an inch thick. Button of bone removed; it was over base of third left

frontal convolution. Complete recovery.—Mynter, *Annals of Surgery*, May, 1894, p. 539.

Case 18.—Male, aged 23. Compound fracture of the skull, and wound of the arm centre. History of blow on the right side of the head; with compound fracture; marked depression and extensive comminution—the depression being at the centre of the Rolandic region. The left arm below the elbow



FIG. 11.

Case 16. Hudson. Showing location of clot.

was completely paralysed. The intellect was unimpaired, and sensation normal.

Operation three hours later. There was found a laceration of the surface of the brain three-fourths of an inch long, with slight loss of substance. After recovery patient had paræsthesia in left arm and leg. He developed hernia cerebri. Three months later the patient was found to have partial paralysis of the left arm and leg. Some time later the author states that examination showed still the presence of anæsthesia of the fingers and some rigidity of the forearm muscles.—Williams, *New York Med. Journ.*, January 9, 1892. "Motion at shoulder almost normal; can flex and extend the elbow and wrist; pronation and supination perfect; can flex fingers, but cannot com-

pletely extend them. All of the fingers of the left hand are partially anæsthetic, but not analgesic. Perception of heat and cold is normal; the tactile sense alone is affected. He picks up articles with difficulty—fumbles with them as a child would. With his eyes shut he cannot pick up a book, and cannot distinguish between a knife and a pencil held in his hand. The persistence of numbness in the fingers has been noted in a number of cases involving injury of the cortical motor area, and would appear to indicate, at least, a very close relation between the centres for motion and sensation. The inability to pick up articles with the eyes closed, and the awkwardness manifested in picking them up at all, seem to indicate faculty co-ordination, although these symptoms may be partly due to anæsthesia of the fingers."

Case 19.—The patient was a girl, aged 15, affected with Jacksonian epilepsy and progressive paralysis of left upper and lower limbs. Operation and removal of tumour the size of a hen's egg from the right central convolutions. Operation followed by paralysis, which greatly improved. Tactile, general, thermal, painful, and muscular sense impressions, which, before the operation, were intact, were affected in a certain measure after the operation over the whole left half of the body except the face.—Albertoni and Brigatti, *Rev. Sper. di Fren.*, xix., January 1, 1893.

Case 20.—Male, aged 18. History of a fall, followed by symptoms of development of cerebral abscess—headache, fever-hebetude. Four weeks after fall Dr. Nason says: "I noticed left facial paralysis and paralysis of the left arm, with impairment of sensation (chiefly tactile sensation) over the paralysed areas. The tongue also deviated slightly to the left when protruded. I thought, too, that the left leg did not move quite so freely as the right. There was no paralysis of the ocular muscles, and the pupils were equal and reacted to light." Operation: Trephining "over lowest third of fissure of Rolando." An abscess was found opening on the lower third of the ascending frontal; about an ounce of pus was evacuated. On the third day after the operation he regained power in the leg; next day, power and sensation in shoulder and arm. Pain in left arm, face still paralysed. At the end of a week some power in left hand and partial return of sensation. Six weeks after operation "patient seems quite well, and but for fingers of hand being a little clumsy has completely recovered." "The mode of recovery from the paralysis goes to prove that the varicous cortical centres are centres for

associated movements and not for individual muscles; also, that sensation and motion are represented together in the central convolutions. It also would tend to prove that the centres for the various associated movements of the arm are placed from above downward in the following order: Shoulder, upper arm, forearm, wrist and hand, the thumb being lowest of all."

Case 21.—A woman, 55 years old. Glio-sarcoma of the corpus callosum. The patient had suffered for two years from some trouble in walking, but she could use her arms. She finally became dull and apathetic, and was admitted to the hospital. There was then a condition of mental dulness, slight facial paralysis on the left side, slight bilateral spastic paralysis with exaggerated reflexes. A very slight hemianæsthesia to pain obtained on the left side.

The *post-mortem* showed a very œdematous brain, with flattening of the convolutions. The two frontal lobes were united on their median surface. Upon the anterior portion of the corpus callosum there was a tumour which measured from one to three centimetres in diameter, and which grew on both sides into the brain substance, so that the total diameter of the tumour was from five to six centimetres. The centrum ovale was somewhat involved, and the observer thinks that the process might have affected the sensory part of the capsule. There was evidently an extensive destruction of the calloso-marginal gyrus on both sides, with practically no anæsthesia.—Redtenbacher, *Ann. Rep. of the kk. Rudolf-Stiftung Hosp.*, 1891, p. 66.

Case 22.—Man, aged 46. Tumour of the brain, involving the central convolutions of the left hemisphere. The patient suffered from local epilepsy, involving the right arm, and finally with a paralysis and anæsthesia of the right arm and leg.

Operation and removal of the tumour the size of a hen's egg from the middle of the two left central convolutions. The tumour was a sarcoma. After removal of the tumour sensation returned, and also some motion.—Carson, *Jour. of Med.*, 1891, p. 31.

Case 23.—Male, aged 42. History: A blow on the head, followed by symptoms of the development of an abscess. Partial paralysis of left side, including face, arm and leg. Sensibility diminished in the left arm. Three days later, complete left hemiplegia, exaggerated reflexes, diminished cutaneous sensibility over arm, face and trunk.

Trephining over the fissure of Rolando. A trocar was plunged downward and forward to the depth of two inches, and six ounces of pus removed. Death followed. No record of autopsy.—Roy. *South. Med. Rec.*, 1890, p. 456.

Case 24.—Male, aged 21. History of intoxication followed by aphasia with symptoms of dulness of tactile sensation in the fingers, the palm and back of the hand, and wrist. The whole of

the right side, however, showed some defect in sensibility to touch, temperature, and pain, as well as of the muscle sense. Still it is stated that there was no ataxia in the right arm or hand. He could carry his right index to the tip of the nose, but he held the pen awkwardly and dropped it repeatedly. The symptoms increased, some paresis and some right hemiplegia developed, and the dulness of sensation increased proportionately.

Operation of trephining over the Rolandic fissure. A large subdural clot was found; this was removed, and the patient got well.

The sensory symptoms are specifically described as follows :

"Sensation (tested with a pin) is somewhat dulled on the whole of the right side. The main dulness is in the fingers,



FIG. 12.

Case 24. Bremer and Carson. *American Journal of Medical Sciences*, February, 1892.

the palm and back of the hand, and the wrist; it is less higher up to the elbow and shoulder, and much less in the face. But, as just stated, the whole of the left side, including the leg, shows a defect in common sensibility. The same is true of the sense of temperature and pain. Passive movements of the fingers of the right side are not so well perceived as those of the left, showing a lowering of the muscle sense. The passive movements of the toes on the right side, however, are correctly stated. There is no ataxia in the right arm or hand; without hesitancy he carries his right index to the tip of the nose, the eyes being closed, and puts with precision the tip of the finger on the point of a pin.

On the fourth day after the operation: "Sensation in right arm still impaired. Sharp points on right arm recognised as

two points one and a-half inches apart, or fingers two inches apart. Unable to feel blowing of breath on hands or arm."

Discharged cured in three weeks.

The localisation of the lesion is given as shown in the accompanying diagram (fig. 12).

Case 25.—Male, aged 60. History of the gradual development of a brain tumour with progressive left hemiplegia, most marked in the left arm. Sensation diminished in both arm and leg; at times both arm and leg would be almost devoid of sensation, at other times hyperæsthesia was complained of. Gradual development of a complete hemiplegia with increasing hebetude, coma and death.



FIG. 31.

Case 25. Showing the localisation of the tumour which was adherent to the dura mater.

Post-mortem.—A tumour, two and a-half inches in diameter, was found involving the dura mater and the cortex of the middle of the precentral convolution and the posterior parts of the first and second frontal.—Barker, *N. Y. Med. Jour.*, June 25, 1892 (fig. 13).

Barker's words are: "Sensation diminished in both arm and leg. Dynamometer showed a loss of one-half in left hand." "The arm and leg became more paretic, sensation more disturbed. At times both arm and leg would be almost devoid of sensation (to pain and touch?); at other times hyperæsthesia was complained of."

An analysis of these cases shows first that injuries and destruction of the motor cortex are accompanied by disturbance of

cutaneous and musculo-articular sensations.¹ Before one can justly interpret the significance of these sensory disorders, we must agree upon some common interpretation of the significance of the tests used in making the examinations. The author's mode of procedure is shown in the following paragraph :—

(1) The tactile sense gives us sensations of touch, impact, pressure, and of modifications due to extent and character of the surface, so that we recognise smoothness, roughness, &c. It is tested by bringing some object carefully in contact with the skin, or by pressure on the surface, or by the drawing rough and smooth objects over the skin.

(2) The localisation of objects touching the skin is done by means of an associative process, by which former experiences in touch are revived. In localising an object the mental process is more complex than in simple appreciation of contact. While not strictly correct psychologically to say that tactile memories are revived, yet that term sufficiently expresses the idea. When a person loses his power of localising a point touched, and thinks it is on the thumb when it is on the middle finger, he has a tactile amnesia.

(3) The muscle-sense is dependent on the existence of sensory fibres in the muscles, joints and tendons. By it we appreciate the position of a limb, its active movements and its passive motions—*i.e.*, those made by another person. These sensations are tested in ways familiar to all, and their defects are shown in incoördination and inability to indicate, determine weights and the position or degree of motion of a limb.

(4) A more complex "sensation" is that known as active touch. It is the process by which one appreciates the nature of an object that is placed in the hand or on the skin. Thus the patient is made to distinguish between different coins, or between a key and a nail, &c. In this process both the tactile and muscular senses are brought into play, and here associative processes are called into play.

(5) Besides these we get thermal and painful sensations, the methods of testing which are familiar.

Now, in looking over the records of cases, it will be seen that the sensory losses most often observed are those in which the patient cannot localise an object, and in which he cannot tell the nature of an object; that is to say, he loses the localisation power and the power of associating muscular and tactile sense, the power of active touch (*Ziehen*). Fumbling and awkwardness are clinical characteristics of destructive lesions of the motor cortex.

It is the associative power which is first and most impaired. After this we find most often tactile anæsthesia, then analgesia, then simple muscular anæsthesia, and least often loss of temperature sense.

¹ The author has not attempted to deal with the negative cases, in which cortical injury occurs without apparent sensory disturbance. The number of such cases is becoming yearly less.

This is in accordance with the evolutionary doctrine that the higher and more complex functions are more differentiated, more localised, and first affected by disease.

The simple tactile sensations are very diffusely represented in the cortex, and small lesions affect them slightly and temporarily, but the localising power, depending on tactile memories, and the "active touch," dependent on motor and tactile memories, are each associated with the special cortical motor centre for the particular part.

In small cortical lesions, therefore, we have (besides the paralysis) a kind of amnesia, which may be compared to the motor aphasia which results when Broca's centre is destroyed.

Naturally, before a set of motor nerve cells is discharged in a voluntary movement this discharge must be co-ordinated and set off in a definite manner. The skin-muscular sensations stored up as memories¹ regulate these voluntary and automatic movements just as the word memories regulate the organs of articulation. In cortical lesions there is then a skin and muscular "amnesia," and the patient with a cortical lesion is practically a case of motor-amnesia, so far as localisation and certain purposeful movements in the affected part are concerned. The application of the same psychology to the central convolutions that is applied to that of Broca solves, as it seems to me, the much-mooted question of the motor-cortex and its functions.

The other and concomitant symptoms of analgesia and thermo-anæsthesia can be equally understood on this hypothesis. Painful and thermic stimuli help in teaching the motor-cortex to discharge correctly and purposefully. Pain and thermic memories would naturally cluster around memories of contact and motion, yet they are less specialised, less important, less effective.

The muscular sensations have their cortical representation, probably in the inferior parietal lobule and its neighbourhood. But the awkward and fumbling movements in lesions of the central convolutions can be explained by the view suggested, that muscular memories or association processes are represented measurably, at least, in the motor area.

The author does not, however, subscribe to the doctrine of Bastian and others, who believe that the central convolutions are sensory centres. They are essentially motor. Nor is it enough to say that the motor-cortex is sensori-motor, for this does not imply the fact that the cortex represents not only sensations, but also memories of sensations. It is, in fact, a sensory-memory-motor organ.²

¹ As already stated, the term memory is used to make the description harmonise with our ordinary terminology in describing the effects of cortical lesions.

² The terms, "perception" or "association-revival" areas are perhaps more strictly correct from the psychologist's standpoint than memory areas. But the word, memory, as ordinarily used by neurologists, answers the present purpose.

BRAIN.

PARTS II. & III., 1896.

Original Articles and Clinical Cases.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.

BY HENRY HEAD, M.A., M.D.,
Medical Registrar to the London Hospital.

PART III.—PAIN IN DISEASES OF THE HEART AND LUNGS.

INTRODUCTION.

IN my previous papers I have dealt with referred pain and superficial tenderness from the topographical standpoint. Where a single area on the body or on the scalp was well marked out, I laid stress on that particular case, oblivious of whether it was typical of the disease which caused it or not. When an organ was associated with a large number of segmental areas, no attempt was made to show the significance of different groupings of these areas in the diseases of that organ.

The following paper treats, not of the topography of the segmental areas, but of the pain caused by diseases of various organs. Thus I lay no weight on the appearance of a single area on the body or the scalp, but attempt to show the meaning of groups of areas of superficial tenderness.

I do not presume to write a treatise on diseases of the heart and lungs, but only to show how diseases of these organs illustrate the distribution and the origin of referred pain and superficial tenderness.

This enquiry is essentially a physiological one with clinical cases in the place of laboratory experiments. Thus many of the cases I shall give as illustrations are not intended to illustrate the natural history of the disease, but simply serve as links in an investigation of the causes which lead to referred pain.

It has been the custom to speak of many of these pains as "intercostal neuralgia," "rheumatic pains," and by other names of a like kind. The referred headaches associated with these pains have been called "megrin," "cardiac headaches," and the like. I hope I shall be able to show that such words are unnecessary and act perniciously by soothing the conscience of the observer.

CHAPTER I.

PAIN IN DISEASES OF THE HEART.

§ 1. *Introduction.*

The occurrence and significance of pain during the course of diseases of the heart has not been systematically dealt with in any of the text books on the subject. Most authors mention certain specific instances, such as the pains associated with aneurism, and those of angina pectoris, but no systematic attempts have been made to note the presence or the absence of pain and the character it assumes in each of the types of cardiac disease.

It is useless to select only those cases which suffer from pain, for the absence of pain is often more instructive than its presence. The presence or absence of pain should be noted in each case as part of the routine examination.

Then we must determine whether if pain be present it is local or referred. In some cases, as when it involves the neck or the arm, it is obviously referred; but in many cases it would be impossible to say, had we only the patient's description to guide us, whether it were referred or not. This is especially the case with those pains which lie in the region of the heart itself.

It, therefore, becomes necessary to examine the superficial structures of the chest, back, and arm to see if they be tender or not. For if superficial tenderness is present the pain which accompanies such tenderness is certainly referred.

Such hyperæsthesia of the skin has long been recognised as an accompaniment of affections of the heart. Thus Hilton,¹ mentions tenderness of the upper part of the chest as a symptom in diseases of the heart and great vessels, and Walshe places it amongst the occasional concomitants of cardiac disease. The first of these observers shows throughout his works how close he was to the truth with regard to referred pain and its accompanying tenderness; but he was misled by his knowledge of the gross anatomy of the peripheral nerves and by the want of that knowledge of the construction of his sympathetic system which we owe entirely to Gaskell's initiative. Walshe, with his splendid powers of observation, evidently suspected that hyperæsthesia might be a symptom of importance; for he does not dismiss it lightly, but notes that its significance is a matter for further research. Since then writers on cardiac disease have entirely neglected superficial tenderness, dismissing it with a slighting reference to hysteria under complete misapprehension of Charcot's teaching as to the nature of his hysterical points.²

Referred pain and superficial tenderness must remain inexplicable so long as we classify cardiac disease according to the name of the valvular defect. For the chief factor in the production of referred pain and its attendant phenomena is some alteration in the tension within the cavities of the heart secondary to the disease of the valves.

Now in some cases of valvular disease the secondary changes within the cavities of the heart are plain; but in many other cases we are ignorant of the conditions within

¹ "Rest and Pain" [4th edition], p. 258.

² Since Mackenzie [*Med. Chronicle*, 1892] and myself independently recognised the significance of superficial tenderness in visceral disease, I only know of the following papers bearing on the occurrence of this symptom in cardiac disease—Mackenzie "Heart Pain" [*Lancet*, Jan. 5, 1895], James [*Brit. Med. Jour.*, June 29, 1895].

the heart under which the circulation is carried on. Our knowledge of these conditions is based on our interpretation of physical signs, and it is in this very interpretation of the same physical signs that the greatest differences of opinion exist. No new physical sign or method of examination has been discovered, and yet our knowledge of the physical signs which point to mitral stenosis has advanced considerably of late. Thus it may happen in the future that the laborious association of certain symptoms with what we suppose to be some one or other condition within the heart may become valueless, owing to an increase in our knowledge of the significance of some particular association of physical signs that has as yet escaped observation.

I shall, therefore, in all cases state the conditions which determine the presence or absence of referred pain in terms of physical signs. Later, I shall devote a chapter to a consideration of what I believe to be the interpretation of these signs, and the bearing of this interpretation on the causes of referred pain. Thus if my interpretation should prove incorrect, I hope that the observed association between the presence or absence of pain and certain conjunctions of physical signs may still remain.

Every case upon which this work is founded has been taken by myself. For in such an investigation it is all important that the reader should have to deal with the errors of a palpable individual, rather than with those of impalpable ghosts. In every case from the Victoria Park Hospital for Diseases of the Chest, the physical signs were checked by other observers more skilled than myself. To Dr. Harrington Sainsbury and Dr. Colbeck, my thanks are particularly due for the trouble they took to insure the accuracy of my physical signs in case of cardiac disease.

My object here is to elucidate the conditions which cause referred pain, and I have therefore excluded all cases from these pages in which competent observers diagnosed compound lesions. For although such compound lesions form very instructive puzzles for a commentary, they can only be explained when we have settled the primary laws by a study of simple lesions.

§ 2. *Local Pain in Diseases of the Heart and Pericardium.*

The pain that is produced by diseases of the heart may be local or referred. Before passing to the characteristics and distribution of referred pain, which forms the most important portion of this chapter, I will touch shortly on some features of the local pain that occurs with certain cardiac lesions.

In examination of every patient it is necessary first to exclude referred pain, and its accompanying superficial tenderness before concluding that the pain is local. Now when the pain is situated over the arm, no difficulty can arise, for such pain in disease of the valves of the heart must be referred and not local. But when the pain lies, at any rate, partly, over the heart itself, such pain is commonly, though in most cases erroneously, supposed to be local and not referred.

Attention to the following points of difference enable us to decide whether the pain be local or referred :—

REFERRED PAIN.

1. Referred pain is not only situated on some spot on the front of the chest, but also over some spot behind at a distance from the cardiac area.

2. Referred pain is associated with more or less tenderness of the coverings of the chest and back, elicited by picking up the skin and subcutaneous tissues between the fingers.

3. This superficial tenderness frequently passes round the body in a more or less horizontal band from the middle line of the back to the middle line in front. If less acute it lies over one or more spots, one of which is situated over the anterior, the other over the posterior aspect of the trunk.

4. Pressure as a rule relieves superficial tenderness.

5. The superficial reflexes are increased over an area of the superficial tenderness accompanying referred pain.

6. Referred pain is frequently accompanied by headache and superficial tenderness of the scalp, obeying rules with regard to its distribution which I laid down in my second paper.

LOCAL PAIN.

1. Local pain is confined strictly to the area of the heart or pericardium, and does not pass round the body or through to the back.

2. Local pain is accompanied by no marked superficial tenderness. But when the intercostal spaces over the painful area are either pressed or percussed, the pain is increased, the amount of tenderness elicited depending on the force applied.

3. When present, deep tenderness can only be elicited over the heart or pericardium, and is absent from the posterior aspect of the trunk.

4. Pressure increases deep tenderness.

5. The superficial reflexes are unaltered over the area of deep tenderness accompanying local pain.

6. Local pain is unaccompanied by that form of headache which is associated with scalp tenderness. Headache if present is a separate phenomenon, standing in no direct relation to the local pain and deep tenderness.

A.—Local Pains in Valvular Diseases.

It is commonly supposed that local pain is far commoner in cardiac disease than referred pain. This I believe to be a pure assumption. Probably local pain is a distinct feature of the majority of painful cardiac states, but it is usually so masked by referred pain that its presence is very difficult to determine.

But in certain cases of mitral disease, especially when the pulse is irregular, the patient complains of palpitation accompanied by pain. This pain is said to lie over the apex beat, and is unaccompanied by superficial tenderness. It seems to be a true local pain. Now this element must exist in other cases in which referred pain is also present; but the presence of this referred pain masks the local pain, and to say that a true local pain is present is but an assumption.

In the same way, in anginal attacks of the suffocative type [*vide* §7], an initial, stretching, bursting pain is felt at the epigastrium, which is undoubtedly local, and is a phenomenon quite apart from the referred pain. Local pain seems to be most often present when the heart is acting irregularly, and is peculiarly associated with a feeling of breathlessness.

Such local pain in the heart is not, however, definitely associated with deep tenderness, but simply lacks all the points which characterise a referred pain.

B.—Pain in Pericarditis.

When the pericardium is acutely inflamed, pain is present, which shows the most striking contrast to referred pain. Like inflammation of the pleura and peritoneum, inflammation of the pericardium produces a true local pain, with more or less marked deep tenderness.

But in the adult, inflammation of the pericardium is frequently associated with marked disease of the endocardium. Thus in adults the true phenomena due to inflammation of the pericardium are frequently masked, and all satisfactory examination rendered impossible by the coincident superficial tenderness due to the endocarditis.

Occasionally, however, the opportunity arises of examining a case in which superficial tenderness is absent. The local character of the pericardial pain and its association with tenderness on pressure and percussion (in the inter-spaces) are then extremely manifest. It is also interesting to see how closely the area of deep tenderness corresponds in some cases with the area over which friction is audible. Case No. 1 shows all these points distinctly, and happened to be one in which all the characteristics of the local pain were demonstrated to the satisfaction of several other observers.

Case 1.—Illustrating the local pain and deep tenderness produced by Pericarditis.—Nellie T. [Marylebone Infirmary, Mr. Lunn], aged 15, servant.

November 16, 1895.—The left knee began to swell and was very painful. She remained in bed two weeks as the doctor said she had rheumatic fever. Towards the end of the two weeks her heart became bad, and she was admitted December 2, 1895.

No previous attacks of rheumatism. Nothing worthy of note in the past or family history.

On admission [December 2, 1895].—The face was pale, with a slight underlying grey look, but no actual cyanosis. No oedema. No jaundice. The right wrist was swollen and painful, but the fingers were unaffected. The left wrist is becoming swollen.

Respiration is rapid with evident subjective dyspnoea. The *alae nasi* are dilating and trembling irregularly. Short, dry, hacking, semi-voluntary cough. Speech interrupted by the shortness of breath. Chest moves with every inspiration, and the respiratory rate is about 50 in the minute.

She complained of pain over the heart, and this pain was much worse when she coughed. The pain lay definitely over an area in the fourth and fifth spaces on the left side, about the size of the palm of the hand. The pain did not go through to the back or radiate round the body. There was no pain elsewhere, and no headache.

The most careful examination failed to reveal any superficial tenderness anywhere. She is a most intelligent girl, clever, and bright beyond her years.

On the other hand, there was exquisitely marked tenderness on pressure and on percussion over an area in the fourth and fifth spaces, about the size of the palm of the hand, and with its centre about in the left nipple line.

Cardiac pulsation is widely visible in the fourth and fifth spaces, and also at the epigastrium.

Cardiac dulness begins above at the upper border of the second rib, extends $1\frac{3}{4}$ in. [4.5 cm.] to the right of the middle line, and 5 in. [12.5 cm.] if not more, to the left of the middle line.

Rough to and fro friction is heard in the fourth and fifth intercostal spaces over an area corresponding very closely to that of the tenderness on percussion and pressure.

Over the apex, and as far inwards as the left sternal border, the first sound is absent, and is replaced by a soft blowing systolic murmur which is not conducted into the axilla. Over the lower part of the sternum, both first and second sounds are audible.

Pulse 124, regular, low tension, compressible with distinct tendency to diastolic.

No capillary pulsation. Pulsation in the veins of the neck.

Beyond some few crackling sounds at the left base, no abnormal physical signs present in the lungs.

Temperature on admission 102. Fell next day to 100.2.

Urine, specific gravity 1020, acid. No albumen.

Three leeches were applied over the area of deep tenderness. The ease they gave was obvious, and, within five minutes from the time they bit, she said she no longer felt the pain, and that her breathing was easier.

In four days all friction had disappeared, but the signs of fluid in the pericardium were exceedingly marked. The cardiac dullness extended considerably more to the right, and as high as the junction of the first rib, with the sternum on either side. The deep tenderness disappeared, and never reappeared throughout the course of the case.

§ 3. *Referred Pain in Diseases of the Aortic Valves.*

I have begun with the pain which appears with lesions of the aortic valves, because from its position over the upper part of the chest and arm, at a distance from the heart, it is most obviously referred. Again, the association of the presence or absence of pain with certain conjunctions of physical signs is striking. And, lastly, the interpretation of these signs is less open to dispute than is the case with lesions of other valves.

Pain of the character I am about to describe, occurs most typically in young people who, as a sequel to acute rheumatism, present the following typical signs: a diastolic murmur over the sternum, conducted more or less towards the apex beat, accompanied by a systolic murmur over the upper part of the chest, conducted into the great vessels, and absence of the aortic second sound. The first sound is present over the apex beat or in the axilla, and no mitral systolic murmur is audible at the angle of the left scapula.

But sometimes an attack of acute rheumatism leaves behind it a loud systolic murmur, heard over the aortic area, followed by a perfect second sound and no diastolic murmur of any kind. The pulse is peculiar neither for the strong stroke and low tension, characteristic of regurgitation, nor for the small stroke and maintained tension which accompanies marked stenosis. Such cases present no marked symptoms, and pain is absent. The lesion of the aortic valves, though acoustically patent, is probably physiologically of little importance.

Pain is also a frequent symptom in those cases of aortic regurgitation which arise without a history of acute rheumatism in men of middle age and laborious occupation. But here again the aortic second sound is absent. No mitral systolic murmur must be audible, and the pulse must be collapsing in character.

When present the pain tends to be situated over the upper three or four intercostal spaces of the left half of the chest, in the upper interscapular region on the left side of the back, and on the inner side of the left arm as far as the elbow. Pain is also sometimes present in the left infra-clavicular region, and over the left side of the neck. This pain is frequently present on the left side alone, but if it is severe it spreads according to the laws I laid down in my first paper, and involves the same parts on the right side of the chest and back, and the inner side of the right arm. In such cases the pain appears later on the right side than on the left, and disappears from the right side earlier than from the left half of the chest.

When severe the pain is said to shoot and dart, but when less severe it is stationary over certain points. Thus the pain on the inner side of the arm to the elbow, when severe, is said as a rule, to shoot upwards from the elbow to the shoulder, but when less severe to be situated over the inner condyle of the humerus.

If the pain in the chest is severe the patient not uncommonly complains of headache, "like neuralgia," situated in the forehead, "over the nose and eyes." This headache comes and goes with the pain in the chest. It is

more marked when the pains in the chest and arm are severe. It is more marked on the left side of the forehead than the right, and is sometimes confined to the left side of the head, in cases where the pain is confined to the left side of the chest and arm. The eyes are said to ache when the headache is present, and not uncommonly the patient becomes unable to read, owing to the pain in the eyes, even when no error of refraction is present. If, however, latent hypermetropia exists, this hypermetropic defect may become manifest under the influence of the headache.

When the sharpness of the pain has somewhat abated, the patient not uncommonly complains that the chest feels sore. On examination, certain areas of the skin of the chest and arm are found to be tender, just as with every other referred pain of sufficient severity and duration. This tenderness of the superficial structures lies mainly over the second, third, and fourth interspaces in front, the inner aspect of the left arm, and an area of the back, situated roughly between the level of the first dorsal and the sixth dorsal spines. The area involved comprises the second, third, and fourth dorsal segmental zones to a greater or less extent. The third and fourth cervical areas are also frequently affected and the pain is then said to extend above the clavicle.

In the same way the headache is associated with distinct tenderness of the superficial structures of the forehead, occupying mainly the frontal, mid-orbital, and fronto-temporal segmental areas of the scalp.

To illustrate these points I give in detail the case of a young girl with marked double aortic murmurs, retention of the faint sound in the left axilla, and no systolic murmur heard at the angle of the left scapula. [Case No. 5, table i., p. 166.] The pain was never very severe, excepting after some particularly indiscreet exertion, to which these aortic cases are peculiarly prone. The tenderness represented [fig. 1] is that usually present when she was not confined to her bed.

*Case 5.—To illustrate the non-paroxysmal referred pain produced by lesions of the aortic valves.—*Louisa I. (Marylebone Infirmary, Dr. Lunn), aged 19, servant.

At the age of 13 she had rheumatic fever, during which the right wrist and left ankle swelled and became painful. She was more or less ill for ten months. About a year afterwards was suddenly taken bad with a pain in the upper part of the left side of the chest and in the neck. In 1892, at the age of 16, she went to service. She then suffered from attacks of shortness of breath, especially after exertion. In 1893 pain became a marked feature, and she was admitted to the infirmary. She much improved, and was discharged, but was re-admitted in 1894. She again improved, and took another situation, but the pain became so bad she was re-admitted in October, 1895.

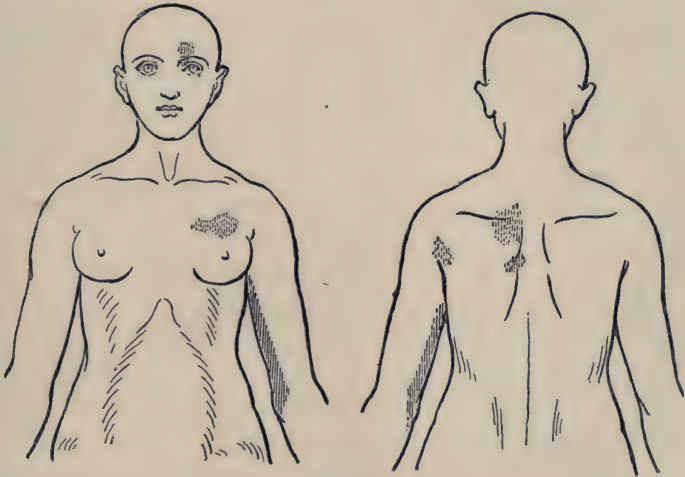


FIG. 1.

To show the areas of superficial tenderness in a Case of Aortic Obstruction and Regurgitation [Case No. 5.]

The area mainly affected is that of the 2nd dorsal segment.

Slight scalp tenderness was present on the left side of the forehead.

December, 1895.—Her condition was as follows :—Very bright, small, slight girl of 19. The greater part of her face is pale, with an irregular underlying vascular mottling. On her cheeks is an irregular pink, mottled flush, which comes and goes. No true anæmia. No œdema. No jaundice. No clubbing of fingers. No nodules. No wasting.

She has no feeling of faintness except with the attacks of pain. When not in bed the heart beats very fast on any exertion, and this seems to bring on the pain. Not short of breath when in bed, but when up and about she has a tight feeling in her chest as if she was going to be choked.

When kept strictly in bed she is free from pain and headache, and all superficial tenderness is absent. When up she is an exceedingly active worker. Pain then appears over the second

left intercostal space, close to the anterior axillary fold, which passes through to the back, close to the vertebral border of the spine of the left scapula. A variable amount of superficial tenderness is present, mainly over the area of the second dorsal segment (fig. 1).

She has occasional headache over the eyes, with slight tenderness of the forehead on the left side (fig. 1).

The pulse, when she is up, is about 100, regular, of good stroke, but typically collapsing. Marked pulsation of the arteries of the neck.

Marked capillary pulsation.

No pulsation of the veins of the neck.

Heart's apex beat in fifth space four inches (10.5 cm.), from the middle line (just outside nipple line). It is heaving. No thrill.

Cardiac dulness beginning at third rib, extends to right edge of sternum, and four and three-quarters inches (12 cm.) to the left (about one inch outside the left nipple line).

Over the whole front of the chest, extending to the mid-axillary line, a loud systolic murmur is heard, followed by a diastolic murmur, forming the typical to-and-fro aortic murmurs. Over the aortic area neither the first nor the second sounds are heard. Over the actual apex beat the first sound is just audible as a dull booming, but on travelling outwards into the left axilla the first sound becomes quite distinct, and is long and booming, in spite of the fact that the aortic systolic murmur is still faintly heard. At the angle of the left scapula a booming first sound is distinctly audible, unaccompanied by any murmur. The pulmonary second sound is audible, but is not accentuated.

Respiration 24, quiet, regular. No cough.

No abnormal physical signs in lungs.

Tongue clean, moist. Appetite good. No pain after food. Bowels irregular.

Urine normal. No albumen.

Vision $\frac{6}{6}$. Jäger 1. No hypermetropia.

Up till April, 1896, the physical signs and main features of the case have remained unaltered.

A marked feature of the pain which appears in aortic disease is the tendency it shows to sudden exacerbations. The pain rapidly spreads, the patient feels exceedingly ill, and, if the spread of the pain is sufficiently rapid and wide, the attack may resemble true primary angina pectoris. Although no hard-and-fast line separates these attacks of increase of pain from the violent anginal attacks which occur in some patients with aortic disease, I shall deal with the whole subject of paroxysmal pain and angina pectoris in a later section [§ 7, p. 202].

All cases of aortic regurgitation, or of regurgitation and stenosis, are liable to suffer from occasional referred pain and tenderness so long as the apical first sound is good and no mitral systolic murmur is heard at the angle of the left scapula. That is to say that so long as the mitral valve holds, pain is liable to appear. If, however, the first sound at the apex and in the axilla is replaced by a systolic murmur conducted to the angle of the left scapula, pain, such as we have learnt to recognise as the possible accompaniment of aortic disease, is absent.

If a patient has long suffered from referred pain and tenderness over the upper dorsal segments, and the mitral valve then fails as a late feature of the disease, it might be said that the pain ceased because the patient's faculties were too low to originate or to appreciate pain.

I have, therefore, collected a series of cases from my notes in which the apical first sound was abolished and replaced by a systolic murmur, conducted to the angle of the left scapula, as a primary feature of the disease. In these cases the mitral regurgitation was not a secondary matter due to heart failure. I have arranged them in a table so that they be compared with a similar table of those cases in which the first sound at the apex was intact, and no systolic murmur was audible at the angle of the left scapula. The absence of referred pain and tenderness in the former group, and its presence in the latter, is striking.

Thus the conclusions to which we have arrived in this section may be summed up as follows:—

(1) When an aortic diastolic murmur, with or without an aortic systolic murmur, is present, the aortic second sound abolished, and, when the pulse shows marked collapsing characteristics, referred pain is liable to form a symptom of the disease.

(2) If in addition to these signs the first sound is absent both over the apex and in the axilla, and a systolic murmur is heard at the angle of the left scapula, referred pain of cardiac origin will be absent. If this condition arises owing to cardiac failure, the pain which was previously present will disappear, and those cases in which this conjunction of

TABLE I.—DISEASE OF THE AORTIC VALVES, WITH A GOOD

No.	SEX & AGE.	APEX BEAT.	THRILL.	AORTIC.			
				1ST.	2ND.	SYST.	DIAST.
2	F. 23	$\frac{6}{\text{Axilla}}$	O	O	O	X	X
3	F. 21	$\frac{5}{1\frac{1}{2} \text{ Extl. N.L.}}$	O	?	O	X	X
4	F. 18	$\frac{5}{2 \text{ Extl. N.L.}}$	O	O	O	X	X
5	F. 20	$\frac{5}{\text{Just Extl. N.L.}}$	O	O	O	X	X
6	F. 50	$\frac{5}{1\frac{1}{2} \text{ Extl. N.L.}}$	O	O	O	X	X
7	M. 40	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	O	X
8	M. 38	$\frac{6}{1 \text{ Extl. N.L.}}$	O	O	O	X	X

TABLE II.—DISEASE OF THE AORTIC VALVES, WITH A MITRAL

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	AORTIC.			
				1ST.	2ND.	SYST.	DIAST.
9	M. 26	O	O	O	O	X	X
10	M. 17	$\frac{6}{2 \text{ Extl. N.L.}}$	Systolic	O	O	X	X
11	F. 42	$\frac{7}{\text{Ant. axilla}}$	Systolic	O	O	X	X
12	M. 35	$\frac{6}{\text{Ant. axilla}}$	O	O	O	X	X
13	M. 43	$\frac{5}{\text{Nipple line}}$	O	O	O	X	X
14	F. 15	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	X	X
15	F. 18	$\frac{6}{2\frac{1}{4} \text{ Extl. N.L.}}$	O	O	O	X	X
16	M. 45	$\frac{6}{2 \text{ Extl. N.L.}}$	O	O	O	X	X
17	M. 38	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	X	X
18	M. 44	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	X	X
19	M. 50	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	X	X
20	F. 30	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	O	X	O

In the third column the figure above the line represents the intercostal space in represents in inches the distance outside the nipple line. Thus $\frac{6}{2 \text{ N.L.}}$ = apex beat in

In the column devoted to the pulse, *regurgit* means that the pulse showed the *Post-mortem Examinations*. Case No. 9.—Aortic valves incompetent; mitral valve valves incompetent; mitral valve admitted five fingers; left ventricle much hyper-three fingers with ease; left ventricle hypertrophied and dilated. Case No. 18.—Aortic cone, 1.55 in.] ; left ventricle dilated and hypertrophied.

APICAL FIRST SOUND AND NO MITRAL SYSTOLIC MURMUR.

MITRAL.			PULM. 2ND.	PULSE.	VEINS OF NECK.	LIVER	REFERRED PAIN AND SUPERFICIAL TENDERNESS.
1ST.	2ND.	SYST.					
X	X	O	X	80 reg. regurgit.	O	+	Sudden attacks of pain. Tenderness Left Cerv. 3 to Dorsal 5. Left shoulder and arm to elbow.
X	X	O	+	80 reg. regurgit.	O	O	Tenderness C ₄ to D ₄ . Mainly Left.
X	X	O	X	80 reg. regurgit.	O	O	Anginal attacks, <i>vide</i> § 7, p. 202.
X	X	O	X	100 reg. regurgit.	O	O	Left shoulder and arm to elbow. Left D ₂ mainly, <i>vide</i> p. 162.
X	X	O	X	84 reg. regurgit.	O	O	L. neck, shoulder and arm to elbow. Tenderness Left C ₄ and D ₂ .
X	X	O	X	100 reg. regurgit.	O	O	Sudden attacks on exertion. Widespread tenderness L. side.
X	X	O	X	100 reg. regurgit.	O	O	Left arm to elbow.

SYSTOLIC MURMUR, ABOLISHING THE APICAL FIRST SOUND.

MITRAL.			PULM. 2ND.	PULSE.	VEINS OF NECK.	LIVER.	REFERRED PAIN AND SUPERFICIAL TENDERNESS.
1ST.	2ND.	SYSTOLIC.					
O	?	Conducted to angle of scap.	X	100 reg. regurgit.	O	O	No pain. No tenderness.
O	Feeble	Conducted to angle of scap.	+	108 irreg. regurgit.	O	O	No pain. No tenderness.
O	Feeble	Conducted to angle of scap.	X	100 reg. regurgit.	Pulsa- tion	+	No pain. No tenderness.
O	O	Heard at angle of scap.	X	96 reg. regurgit.	O	O	No pain. No tenderness.
O	O	Heard to mid. axilla	X	92 reg. regurgit.	O	O	No pain. No tenderness.
O	X	Heard all over the back	+	120 reg. regurgit.	O	O	No pain. No tenderness.
O	2	Heard all over the back	+	96 reg. regurgit.	O	O	No pain. No tenderness.
O	X	Conducted to angle of scap.	X	80 reg. regurgit.	O	O	No pain. No tenderness.
O	X	Conducted to posterior axilla	X	68 reg. regurgit.	O	O	No pain. No tenderness.
O	X	Conducted to angle of scap.	X	84 reg. regurgit.	O	O	No pain. No tenderness.
O	O	Conducted to angle of scap.	X	80 reg. regurgit.	O	O	No pain. No tenderness.
O	X	Heard all over the back	+	88 irreg. lowtension	Pulsa- tion	O	No pain. No tenderness.

which the maximum pulsation of the apex is seen and felt. The figure below the line 6th space, 2 in. outside the nipple line.

low tension collapsing character typical of marked aortic regurgitation.

admitted five fingers; left ventricle hypertrophied and dilated. Case No. 10.—Aortic trophied and dilated. Case No. 11.—Aortic valves incompetent; mitral valve admitted valves incompetent; mitral valve admitted five fingers [diameter, measured with the

physical signs exists from the beginning will not suffer from referred pain.

(3) The pain, when present, is mainly over the upper part of the chest and back, on the left side and in the left arm. It is accompanied by superficial tenderness over the second, third, and fourth dorsal, and sometimes the third and fourth cervical segmental areas.

(4) Headache over the frontal region is liable to be present. This is accompanied by tenderness over the frontal mid-orbital and fronto-temporal segmental areas on the scalp.

§ 4. *Pain produced by Aneurisms of the Aorta.*

The pain produced by an aneurism of the aorta is usually said to be due to: (1) pressure upon the walls of the chest and the vertebra, or (2) pressure upon nerve cords or roots. Now there is no doubt that these factors play a part in the production of the pain, especially when the aneurism has reached a great size. But in the case of smaller aneurisms, such pressure on surrounding structures plays a comparatively small part in the production of the pain. And yet pain is comparatively more common with the smaller aneurisms than with those of great size.

Now a careful examination shows that the pain is accompanied by superficial tenderness in a considerable proportion of cases of aneurism of moderate size. Moreover, this tenderness marks out areas on the surface of the chest and arm, which are to a great extent the same as those I have described above as concomitant phenomena of the pain of aortic disease. Thus this pain, accompanied as it is by superficial tenderness, is a simple referred visceral pain. For there can be no question of pressure on bones or nerves when the same pain arises as a sequel to aortic disease. And yet both aneurisms and aortic disease may cause pain accompanied by superficial tenderness over almost similar areas. We cannot wonder at this when we remember that etiologically the first part of the aorta is as much part of the heart as the ventricle.

The pain produced by aneurisms of the aorta must therefore always be considered under the following three groups:—

(1) True referred pain accompanied by superficial tenderness of the chest, neck, and arm.

(2) Referred sensations due to pressure on nerve trunks.

(3) Local pain produced by pressure on such structures as the vertebræ and ribs.

The commonest situation for the pain is down the inner side of the arm. Sometimes it only extends to the inner condyle of the humerus, but occasionally and not infrequently it extends over the ulnar side of the forearm to the little finger.

It is more common in the left arm than in the right. I cannot help thinking that if the pulsation of the aneurism appears to the right of the sternum, the pain will radiate down the right arm, whilst if it is visible to the left of the sternum, the left arm will be affected. I have not, however, at present sufficient material to make certain of this statement.

The pain is usually of a dull aching character, relieved by pressure or by rubbing. Thus patients who suffer from this pain frequently make the following characteristic movements: The left arm is somewhat adducted so that the elbow lies over the cardiac area of the chest. The right palm is placed over the left arm, so that the fingers reach over to that aspect of the left arm which is now outwards.¹

The right hand is then rubbed up and down over the outer part of the left arm.

Occasionally the pain is of a shooting character, and in that case it usually is said to shoot up from the elbow to the chest and back. If it extends to the little finger it shoots up from that finger to the elbow.

The pain sometimes assumes a paroxysmal character, and secondary anginal attacks may be developed. These are considered in § 7.

The pain is accompanied or followed by more or less marked superficial tenderness over the inner side of the arm, and sometimes over the ulnar side of the forearm to the little finger. When carefully marked out these areas

¹ Owing to the rotation of the left arm which accompanies the adduction this portion which now lies outermost is in reality the inner aspect of the arm.

correspond either to the maxima or to the full extent of the second and first dorsal segmental areas.

Now I pointed out in my first paper (p. 33) that the second dorsal area had a limb on the anterior surface of the chest, over the second space and third rib, and a dorsal limb, characterised by a spot close to the vertebral border of the spine of the scapula. In the same way the first dorsal area has a small portion on the back, at the level of the seventh cervical and first dorsal spine. Thus, if the whole of both these areas are represented, pain and tenderness will be present in the second space in front, and over the first and second dorsal spines, and the inter-vertebral groove to the left or the right of them.

Now in many cases this pain has been mistaken for a local pain, and has been ascribed to erosion of the chest in front, and of the vertebræ behind. But examination of the chest shows no signs of an aneurism of sufficient size to cause erosion of both the anterior and posterior walls of the chest. Moreover, the presence of the superficial tenderness shows that these spots are nothing more than the anterior and posterior limbs of segmental areas, of which the lateral limbs are to be found on the inner side of the arm.

But, it might be objected, that this pain down the inner side of the arm is of itself nothing but the expression of pressure on nerve trunks. But if this were so, it is difficult to see why it should appear in diseases of the aortic valves, following acute rheumatism (Case No. 5, p. 162). Again, pressure upon nerve trunks produces numbness and tingling, but not superficial tenderness, as anyone can learn who will compress his ulnar nerve and observe the result.

Moreover, the distribution of the hyperæsthesia is not that of any peripheral nerve or cord of the brachial plexus. Such a case as No. 21 (p. 171) is entirely incompatible with the supposition that this pain down the arm, to which I now refer, could be due to pressure upon nerve roots.

A most interesting motor phenomenon is occasionally associated with pain and superficial tenderness of the areas on the inner aspect of the arm and forearm. The patient complains that the hand feels stiff and numb. The grasp of

the hand is undoubtedly weaker when the pain is present than when it is absent. Thus I have seen a patient, in whom this pain came on owing to the effort of sitting up to eat his dinner, drop his fork from his left hand. When carefully tested, the movements of the fingers of the left hand were clumsy. There was no absolute paralysis, and no profound paresis that could be localised in any particular muscle, but yet all the fine movements of the left hand were badly performed. Movements of the thumb do not seem to

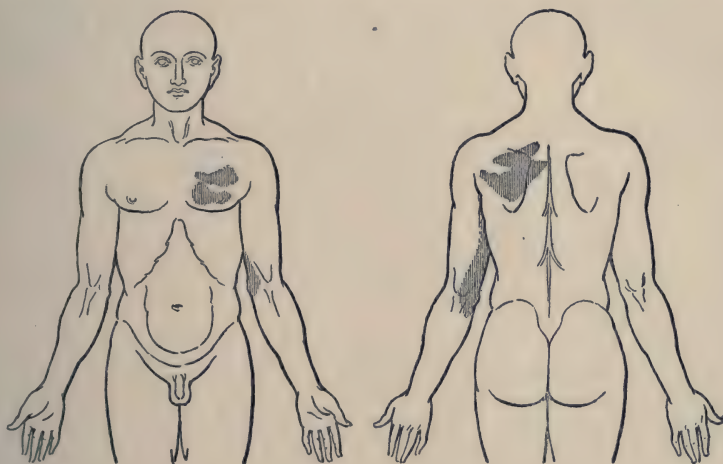


FIG. 2.

To show the superficial tenderness present in a Case of Aneurism of the Ascending Portion of the Aorta [Case No. 21].

The areas mainly affected belong to the 2nd and 3rd dorsal segments.

suffer to the same extent as fine movements of the fingers. This is a very transitory phenomenon, and soon disappears when the pain has gone. The interest of this motor condition lies in the fact that the first dorsal root with the eighth cervical supplies the small muscles of the hand. Thus an extreme disturbance on the sensory side, within the first dorsal segment, seems to cause some motor weakness within the area supplied by the first dorsal and eighth cervical root.

Case 21.—To illustrate referred pain produced by aneurism of the ascending aorta.—Charles T. (V.P.H., Dr. Eustace Smith). Aged 31, carpenter. Admitted, December 8, 1893.

An extremely strong healthy man till seven years ago, when he had a "hard chancre." Was treated with mercury. No secondary rash or other sequelæ.

Two years ago began to suffer from pain in the chest. He is a carpenter, and the pain came on particularly after using the heavy plane. September 22.—The pain became so bad he gave up work.

On admission.—He is a very finely built man of six feet. Face of a uniform pinkish colour, with no flush. No anæmia, jaundice, or œdema. Well nourished.

He is conscious of the pulsation of the swelling. No palpitation or fainting.

He states that three weeks before admission he suffered from violent pain in the left arm, which began in the second phalanx of the little finger and travelled upwards to the inner side of the elbow. The pain then extended as an intense aching over the whole of the ulnar half of the left forearm and in the little finger. Occasionally it darted and shot, and sometimes darted from the inner aspect of the elbow to the joint between the first and second phalanges of the little finger. This pain disappeared a week before admission, and has given place to the pain from which he now suffers. Pain is now present over the inner side of the arm as low as the fold of the elbow. Its point of greatest intensity is situated one inch (2.5 cm.) above the inner condyle of the humerus.

Some distinct superficial tenderness is present over an area extending upwards from the internal condyle of the humerus over the inner aspect of the left arm.

Visible pulsation in the first and second intercostal spaces. In the second space not only pulsation but a distinct systolic thrill can be felt.

The apex beat can be felt in the fifth space in the left nipple line. Impulse is feeble.

There is no bulging of the chest wall over the left front, but the percussion note is high pitched, and there is increased resistance over the upper part of the chest on the left side, extending from the first intercostal space to the fourth rib, and laterally from the left sternal border almost to the anterior axillary fold.

The cardiac dulness extends from the nipple line on the left to the mid-sternum on the right. Above, it merges into the dulness over the aneurism.

A loud systolic murmur is heard over the upper part of the chest on the left side, with its maximum in the second intercostal space. It is followed by an accentuated second sound.

Over the apex the first sound is weak, and is followed by a very soft systolic murmur, which disappears in the left axilla, but increases in intensity along the left border of the sternum to reach its maximum over the area of pulsation. Thus this murmur at the apex seems to be the conducted aneurismal murmur.

No diastolic murmur heard anywhere.

Pulse 80, regular. Artery large and full between the beats. The pulses seem to be equal.

No dyspnoea. No cough. Voice good. Larynx normal.

No abnormal signs in the lungs.

Tongue clean, appetite good. Bowels opened daily.

Liver not enlarged.

Urine 1020, acid. No albumen.

Pupils equal; react to light and accommodation. Both pupils dilate to shade.

December 13.—The pain extended down the ulnar side of the forearm to the little finger for a few hours.

December 14.—It is now represented by pain over the inner condyle, with definite superficial tenderness of inner aspect of arm.

The pulsation is decidedly more marked.

He was put strictly to bed, and the pulsation distinctly diminished, so that by March 5, 1894, the pulsating area in the second space could be covered by the top of the thumb.

Pain was practically absent throughout this period.

March 7.—He again began to complain of pain down the inner aspect of the arm, and marked superficial tenderness was present over the second dorsal segmental area. The pulsation is now visible over an area of three inches by one and a half inches (7.5 cm. by 3.75 cm.).

March 31.—The pulsating area has again increased, and has returned to the size on admission. The pulsation is accompanied by a marked systolic thrill, and the closure of the aorta semilunar valves can be felt over the pulsating area as a sharp slap. Apex beat and cardiac dulness as before.

Over the whole left side of the chest, as low as the fifth rib, a greatly accentuated second sound is audible. The systolic murmur is more widely heard over the upper part of the chest. No murmur or second sound audible in the back. Over the apex beat the first and second sounds are good, and are not obscured by the systolic murmur.

He now complains of pain—(1) over a spot just internal to the nipple in the third space. He places a single finger over this spot, and says that when the pain gets bad it enlarges. This pain passes through to a point in the back in the interscapular space, at a level of about the fourth dorsal spine. He says it is exactly as if someone were cutting him with a knife.

He also has some pain over the second rib, extending through to a point behind, close to the vertebral border of the spine of the scapula.

(2) He also has a dull aching "numb" feeling over the inner aspect of the left arm and ulnar aspect of the left forearm, to the ulnar side of the wrist, ulnar border of the hand, and to the head of the basal phalanx of the little finger. He is exceedingly precise in his description of the limits of this pain. When this

pain is bad he has loss of power in the fingers of the left hand, and very slightly in the thumb. The fingers feel "silly and useless." He could not grip his fork at dinner.

There is absolutely no loss of sensation to touch, pain, heat, or cold, but there is marked and definite superficial tenderness (hyper-algesia) over the areas in fig. 2.

Pulses equal. Pupils dilate to shade, and are equal. Larynx normal.

But with aneurism of the aorta, pain makes its appearance over other regions than these. Pain is sometimes situated in the left shoulder joint and in the supra-clavicular fossa and back of the neck. This pain may be accompanied by superficial tenderness over the third and fourth cervical areas as in the following case (No. 22). Now it is quite impossible that this pain above the clavicle could be due to pressure on nerve cords or roots, and yet, in this case, we see the pain and tenderness over the second and third dorsal merging imperceptibly into that above the clavicle.

Case 22.—To illustrate the pain and tenderness over the cervical areas produced by aneurism of the aorta.—James C. (V.P.H., Dr. Thorowgood), aged 43, sawdust dealer.

I first saw him December 11, 1893. He had suffered from pain in the back for six months. Six weeks before admission a cough came on, and the pain became much worse. He denies gonorrhœa and syphilis. Married, but wife has never been pregnant. No general illness. Not intemperate (two to three pints of beer daily).

On admission he complained of pain above and outside the left nipple, darting through to a point just below the spine of the scapula behind. He also had pain down the inner side of his arm to the elbow. Beautifully marked superficial tenderness was present over these spots.

The physical signs were those of aneurism of the aorta (see Part II. of this work, BRAIN, 1894, p. 371).

The right pulse was stronger than the left.

The pupils were normal and equal. Larynx unaffected.

He left the hospital free from pain, but on returning to work the pain and tenderness returned. But now the tenderness had spread to the fourth cervical area in the supra-clavicular fossa (*vide* fig. 16, p. 371, BRAIN, 1894).

Since this time he has frequently attempted to return to his somewhat heavy work, and each time the pain returned.

March 7, 1896.—The pain is distinctly increasing somewhat. It is still confined entirely to the left side of the chest, shooting through from the scapula to the upper spaces in front, from the

back of the neck to the left shoulder, and down the inner side of the arm to the elbow.

There is beautifully marked superficial tenderness as in fig. 3.

He complains of headache over the left half of the forehead, and this headache is accompanied by scalp tenderness, as in fig. 3.

No fainting. Occasionally feels faint and has to sit down. No marked palpitation. Becomes very short of breath on exertion.

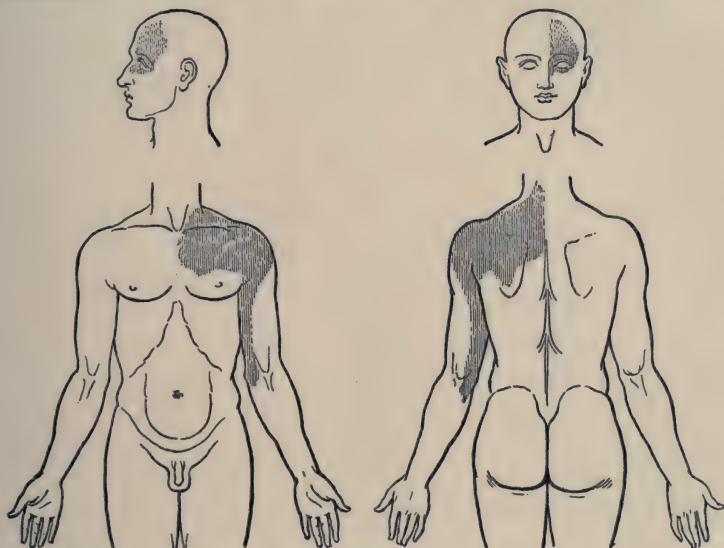


FIG. 3.

To show the superficial tenderness present in a Case of Aneurism of the Aorta [Case No. 22].

The areas mainly affected are those of the 4th cervical and 2nd and 3rd dorsal segments.

Tenderness of the scalp was present on the left side only, over the frontal, mid-orbital, and fronto-temporal areas.

Pulse 64; left pulse decidedly weaker than the right, all up the arm. No pulsation in the neck.

Cardiac pulsation in fifth space and left nipple line ($4\frac{3}{4}$ in. from middle line of sternum). Not heaving. No other pulsation visible or palpable over the chest.

Slight dulness or diminution of resonance over the upper part of the sternum. Cardiac dulness extends from left nipple line to right edge of sternum.

Over the upper part of the sternum and in the second and third right interspaces, a loud accentuated second sound is audible. This second sound is doubtfully heard to the left of the

second dorsal spine behind. A loud systolic murmur replaces the first sound over the aortic area. It is conducted down the left edge of the sternum. At the apex the first and second sounds are good. No murmur heard in the left axilla or at the angle of the left scapula. No tracheal tugging. Larynx normal. Pupils normal.

The same dulness at the right base to the angle of the right scapula, with absent breath-sounds and vocal resonance. These signs have remained unaltered since December, 1893.

This brings me to a point of much theoretical interest and importance. Some cases of aneurism, I believe mainly those which involve the arch of the aorta, complain of pain in the throat. This pain is described as "a kind of cramped feeling," "a sort of painful stiffness," and is situated in the space between the anterior border of the sterno-mastoid and the middle line on one or both sides. This pain is accompanied by superficial tenderness over an area which I have called the inferior laryngeal, an area which is shared by the structures of the larynx proper (fig. 4). Case No. 23 shewed this pain and superficial tenderness to a marked degree. I believe it is particularly liable to occur in those cases in which either the cervical sympathetic (shown by the pupil) or the recurrent laryngeal, or both nerves are affected, that is to say, where there are signs pointing to the arch of the aorta as the situation of the aneurism.

Case No. 23.—To illustrate pain and tenderness over the areas of the front of the neck in aneurism of the aortic arch.—Jane R. (Marylebone Infirmary, Mr. Lunn), aged 53 years, married, charwoman. Admitted November 17, 1895.

October, 1894.—She had a cough and some pain in the back. This did not disappear, and in January, 1895, she went to the Brompton Hospital and was admitted. Thence she was transferred to the Middlesex Hospital, where she remained till her admission to the Infirmary. Throughout this time she has suffered from more or less pain. When she got up the pain grew worse; when she lay in bed it was easier.

Menstruation ceased twelve years ago. Married at 21. Never pregnant. No miscarriages. Never any discharge from the vagina.

Temperate. No history of acute rheumatism or any illness except, possibly, typhoid fever.

On Admission.—She is a well-built, bright, cheerful looking woman of 52. Looks somewhat older. No anæmia. No jaundice. No œdema. Is not wasted.

She says that her heart beats very fast on exertion, but never seems to stop or beat irregularly. This rapid beating is not accompanied by any pain. No shortness of breath whilst in bed. She can lie flat at night.

She has no pain now, and there are no signs of any definite superficial tenderness. No headache.

Pulse 68, regular. Rather small stroke but of well maintained tension. The arterial wall is a little hard. The right pulse is if anything a little stronger than the left.

Apex beat cannot be definitely localised. No thrill. No obvious pulsation seen or felt over the upper part of the chest.

Cardiac dulness begins in the third space, extends $3\frac{1}{4}$ in. (8 cm.) to the left (left nipple line), and to the middle of the sternum on the right. No dulness elsewhere over the chest.

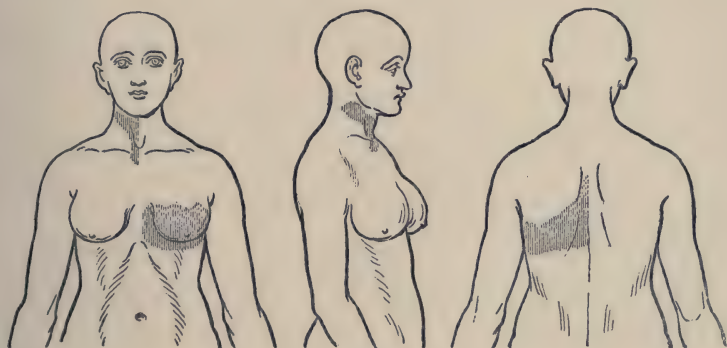


FIG. 4.

To illustrate the pain and tenderness over the Inferior Laryngeal Area of the Neck sometimes present in Aneurism of the Arch of the Aorta [Case No. 23].

Superficial tenderness was also present in this case over the 5th and 6th dorsal areas on the left side of the chest and back.

Over the upper part of, and especially to the right of the sternum, an accentuated slapping second sound is heard. Over almost the same area a soft blurring systolic murmur is audible which is conducted slightly towards the apex. The first sound over the aortic area is feeble. Over the apex the systolic murmur is just audible, and the first and second sounds are good. Neither the second sound nor the systolic murmur are heard anywhere in the back.

No cough now. No marked abnormality in the physical signs of the lungs.

Liver not enlarged.

The voice is a little hoarse, no tracheal tugging. The left vocal cord does not move so well on phonation as the right.

The left pupil under ordinary circumstances is almost 3 mm. in diameter, and is equal to the right. It practically does not react to light or dilate to shade. With a strong light the difference between the pupils becomes very marked. The left pupil dilates somewhat, but not to the extent of the right, under the influence of cocaine. Both pupils react well to accommodation. There is no marked difference in the size of the two palpebral fissures or in the projection of the eyes.

Urine normal.

She remained absolutely in the above condition till the afternoon of January 14, 1896. She then suddenly began to scream with the pain in the head and abdomen. She complained of pain across the upper part of the abdomen on the left side, and over the back below the left shoulder blade. Intense widespread superficial tenderness of chest and back, more marked on the left side. Very marked tenderness over the frontal, mid-orbital, fronto-temporal and temporal areas of the scalp.

Pulse 64, with the same doubtful difference between the two sides.

There is now a distinct fulness over that part of the first and second intercostal spaces on the right side, which lies on each side of the second rib cartilage; thus the second rib cartilage has the appearance of being bulged. A systolic impulse and at times a systolic thrill is felt over this area, followed by a shock corresponding to the second sound.

A loud systolic murmur is heard over the second right rib cartilage, followed by a loud bell-like second sound.

The cardiac signs as before. No sign of dilatation of the left ventricle.

Nothing heard elsewhere in the chest.

The left pupil is markedly smaller than the right, and does not dilate at all to shade.

From this point onwards the pain and superficial tenderness lay mainly within the fifth, sixth, and seventh dorsal areas on the left side. (Fig. 4.) The headache and scalp tenderness were more temporal than before.

March 11.—She still has the pain and tenderness in the left side, but she has just begun to complain of a fresh pain on the right side of the throat. She places her right hand flat over the right side of the space between the anterior borders of the sterno-mastoids, the fingers almost touching the lobule of her ear, and the ulnar border of the hand parallel to the lower border of the jaw. She complains that it is an aching pain like the pain on the left side of the back.

There is more exquisitely marked superficial tenderness over the right inferior laryngeal area (*vide* fig. 4). On the left side of the trunk is seen the tenderness which has been present ever since the attack on January 14.

Occasional slight cough. No alteration in the voice. No marked difference in the movements of the two vocal cords. No

intra-laryngeal cause for the pain discoverable. Cords a normal colour.

Pulse of small stroke. The right pulse is slightly stronger than the left.

There is slight but distinct dulness in the first and second spaces on the right side. Over this area a distinct systolic thrill can be felt. This dulness joins with the upper limit of cardiac dulness across the sternum.

The same systolic murmur and markedly accentuated second sound are audible over the upper part of the right chest.

The left pupil acts to accommodation, but does not dilate in the least to shade.

She became freer of pain, but slight pain and superficial tenderness were still present over the right infra-laryngeal area on April 13, 1896.

The pain of aneurism usually improves greatly under rest and treatment, and all pain and tenderness may disappear after a time. But these cases are notoriously disappointing. Physician and patient are full of hope, when, without obvious cause, or perhaps in consequence of some increased exertion, the pain suddenly returns, not uncommonly with great violence. Suppose the pain and tenderness lay originally over the upper areas of the chest and down the inner side of the arm; with a sudden return of this kind, the pain and tenderness may no longer lie over the upper part of the chest, but may occupy areas under the heart, in the epigastrium and below the shoulder blade (case 23). The tenderness, which previously lay within the fourth cervical and first and second dorsal segments, may now occupy the fifth, sixth or seventh dorsal.

Now we cannot suppose that a new aneurism has suddenly sprung up four inches lower down in the region of the diaphragm, which is above the level of the new outburst of pain. Moreover, the superficial tenderness shows that the pain is referred and not local. Therefore I think we must assume that that portion of the aorta which causes referred pain over the first and second dorsal segments and over the inferior laryngeal area, lies close to that part which causes referred pain over the fifth, sixth and seventh dorsal segments. Now this distribution of pain and tenderness seems to me peculiarly liable to be present in those aneurisms which involve the cervical sympathetic or the recurrent laryngeal,

that is to say, aneurisms of the arch. I shall show reason to believe that the aorta beyond the position of the ductus arteriosus refers into the mid-dorsal region, whilst the aortic bulb and arch refer on to the higher dorsal and inferior laryngeal areas.

Some aneurisms are painless from the beginning or become so in course of time, and many observers have commented on the absence of pain in aneurisms sufficiently large to cause marked symptoms of pressure on the trachea and bronchi. I give as an instance of such a condition case No. 24, where, though there was a history of pain, no referred pain or tenderness was present during the ten weeks he was under observation.

The explanation of this absence of referred pain and superficial tenderness seems to me to be as follows:—The walls of such aneurisms are composed of matted connective tissue, and all true aortic wall has long ago disappeared. The aorta is markedly atheromatous and it is these atheromatous patches which yield to the pressure within. Connective tissue becomes thickened around the yielding patch on the outer side of the aortic wall, and thus the aneurism gradually grows, not by a true distension of the aortic wall, but by the stretching of a wall composed of connective tissue. Thus the wall of the organ, the integrity of which is necessary for the production of referred pain, is no longer present, or is only present in such a condition that the nerves are long past transmitting afferent impulses to the central sympathetic system. Thus such an aneurism may cause local pain by pressure on the structures within the chest, or pain and anæsthesia in the course of nerve cords or roots, but will not cause referred visceral pain accompanied by superficial tenderness.

Case No. 24.—To show how an aneurism that causes marked pressure symptoms may be painless.—William M. (V. P. H. Dr. Eustace Smith) age 53, an engine fitter.

In 1892 he began to suffer from constant pain in the right axilla. This gradually died away. In February, 1893, after all pain had died away, he began to suffer from shortness of breath which gradually became constant. He then began to cough, and

during the summer of 1893 noticed that his expectoration was occasionally streaked with blood.

At the age of 23 he says he had syphilis. No secondary symptoms. Has drunk to excess occasionally. No gout. No rheumatism.

On Admission (February 7, 1894).—Medium-sized man of 53. Face pale and blotchy, with dilated vessels on the cheeks but no true flush. Lips and ears a little blue. Fingers and toes much clubbed. Lower part of the back œdematous.

He complains of palpitation, but only on exertion. It did not cause pain. Short of breath even when in bed; he cannot sleep except when propped up.

He complains of a pain on exertion situated over the epigastrium. He has no other pain. No pain in the arm or upper part of the chest. There is no superficial tenderness anywhere, not even over the situation of the epigastric pain. No headache. No scalp tenderness.

Pulse 88, regular, fair stroke and good tension. Arterial wall distinctly hard. Pulses equal. No capillary pulsation. No venous pulsation in the neck.

General heaving impulse in fourth and fifth spaces. No definite apex beat. No pulsation visible elsewhere. No thrill.

Cardiac dulness much diminished. No dulness over the upper part of the chest.

Over the upper part of the sternum and to either side of it is heard a low-pitched systolic murmur. This murmur is conducted down the sternum, but is not heard at the apex of the heart. It is heard in the left interscapular fossa and over the spine with greater intensity than in front. The aortic second sound is heard but is not increased. Pulmonary second sound short and sharp. At the apex first sound is booming, second sound good.

Respiration about 30. Distinct dyspnoea. Alæ nasi dilating and sterno-mastoids acting. Harsh, dry, tearing cough, more troublesome at night. He brings up a small quantity of tough, aerated mucous expectoration.

Chest somewhat rigid. Movements very poor at both bases, but about equal on the two sides. Hyper-resonant note all over the chest. Breath sounds and vocal resonance good at apices but weak over both bases. Moist râles over both bases.

Tongue red, fairly clean and moist, no pain after food now. Bowels confined.

Liver can just be felt. Spleen not felt.

Urine, sp. gr. 1025, acid. No albumen. No sugar.

He had a very irregular temperature, rising sometimes to 102° F. (39° C.), but frequently not reaching 100 as the highest point in the day. He began to spit up small round masses of mucopurulent material in considerable quantity, and signs first of consolidation and then of excavation began to appear over the right lung. By April 15 the whole right side was implicated, and he died on April 18.

Throughout the whole of this time he suffered from no pain, except that at the epigastrium. No referred headache or tenderness.

Post-mortem.—A sacculated aneurism was found about the size of a large orange. It began below, about $\frac{3}{4}$ in. (2 cm.) above the aortic valves, and involved the greater part of the transverse arch of the aorta, including the origin of the innominate and left carotid arteries. The right branch of the pulmonary artery was extremely flattened and seemed to be running in the substance which formed the wall of the aneurism. Its lumen was almost completely occluded. The left branch of the pulmonary artery was stretched over the front of the aneurism, and its lumen was diminished but not occluded. The aneurism was filled with laminated clot and its walls showed no recognisable remains of the normal aortic lining. They were formed of tough more or less uniform connective tissue. Where not affected by the aneurism the aorta was intensely degenerated. The wall contains much calcareous matter, and in places the coats are infiltrated with blood. Right ventricle much dilated and hypertrophied. Left ventricle small, normal. Aortic valves slightly atheromatous but competent. Mitral valves normal.

Right lung contained one large ragged walled cavity filled with blood-stained stinking grumous fluid. The greater part of the lung was gangrenous, apparently from occlusion of the right pulmonary artery. Left lung normal.

To sum up the conclusions to which we have arrived in this section:—

(1) Aneurism of the aorta may cause referred pain, associated with superficial tenderness over the following areas:—(a) Third and fourth cervical and first, second and third, and occasionally fourth, dorsal segmental areas usually on the left side but occasionally on the right side. (b) Over the inferior laryngeal area on one or both sides. (c) Over the areas from the sixth to the eighth dorsal. These areas appear when we have reason to suppose the aneurism has spread beyond the point where the ductus arteriosus enters the aorta.

(2) Aneurism of the aorta may cause local pain from pressure on structures in the chest other than nerves.

(3) Aneurism of the aorta may cause pain, and occasionally, though rarely, anæsthesia from pressure on peripheral nerves or roots. Such pain is not associated with superficial tenderness over segmental areas. Contrary to the

usual opinion this is the least common cause of pain in aneurism.

(4) Aneurisms which arise in a highly diseased aorta (case 24) or such as are bounded by matted connective tissue only, cause only local and no true referred visceral pain.

§ 5. *Referred Pain in Disease of the Mitral Valves.*

There are no lesions about which greater difference of opinion exists than those of the mitral valve. For at one time the presence of a systolic murmur over the apex in organic cardiac disease at once led to the diagnosis of mitral regurgitation. That the murmur is due to some regurgitation through the mitral orifice is highly probable, but we want to know whether it is the main lesion or not. Broadbent and Graham Steell¹ have long taught that this murmur alone tells us very little with regard to the condition of the mitral orifice, and that the important fact is the relation of the murmur to the first sound of the heart. Thus the only sign of marked mitral stenosis may be a systolic murmur accompanied by a sharp first sound at the apex. In such a case the murmur is that of regurgitation, but the important pathological factor in the physics of the circulation is stenosis of the mitral orifice.

In the course of this research I soon found that it was quite impossible to classify my observations on the referred pain that occurred with lesions of the mitral valves according to the name of the supposed valvular lesion. For in any one case competent observers would disagree in how far regurgitation or stenosis were the main factor. Now the presence or absence of referred pain depends on the conditions within the cavities of the heart. As these conditions can only be inferred from a knowledge of the conditions of the valves, such fundamental differences of opinion amongst experts formed a severe hindrance to me in the earlier stages of this work. With regard, however, to the actual physical signs, I found no such difference of opinion; the difference lay rather in the interpretation of these signs.

¹ Broadbent, *American Journal of Med. Sciences*, No. 181, n.s., Jan., 1886. Graham Steell, *Med. Chronicle*, May, 1888; Sept., 1895.

Throughout this section I shall therefore speak as little as possible of mitral stenosis or mitral regurgitation, but shall attempt to show that, with lesions of the mitral valve, the presence of certain conjunctions of physical signs is associated with referred pain, whilst when certain other physical signs are present, pain is absent. My ideas of the significance of these physical signs are relegated to a special theoretical chapter (chap. ii.).

By adopting this plan of speaking in terms of physical signs I hope that my observations will stand the test of time, though my ideas of the significance of these signs may be falsified by future research. I am certain that if this plan had been adopted by all writers on the subject of mitral disease, much of the present confusion would have been avoided.

In this section I shall only treat of those cases in which the right heart has not failed. For, as soon as the liver begins to be enlarged, pain may make its appearance which is not directly due to the heart. Such cases are reserved for § 6. Thus the auscultatory signs which most concern us here are the mitral diastolic, præ systolic, and systolic murmurs, and the conditions of the apical first and second sounds. Of the diastolic and præ systolic murmurs nothing need be said, but a few words are necessary about the systolic apex murmur.

A systolic murmur heard at the apex may weaken or abolish the first sound, so that the first sound may be absent over the apex, in the axilla, and at the angle of the left scapula. On the other hand, the same murmur may be accompanied by a short, sharp, first sound, such as is usually heard in conjunction with a præ systolic mitral murmur. As Steell¹ has pointed out, a systolic murmur of this kind, associated with a sharp, first sound, is usually not conducted into the axilla, or heard at the angle of the left scapula. But he recognises that this conduction or non-conduction to the angle of the left scapula is an unsafe guide. Thus the real point of importance is not the conduction of the murmur, but the fact that it is associated with a sharp

¹ *Med. Chronicle*, May, 1888, and Sept., 1895.

first sound, either at the apex or in the axilla. That these observations of Broadbent and Steell are of vital physiological importance, and not simply of academic interest, is borne out by the value of these signs as a basis for the classification of a physiological condition such as referred pain.

(a) Whenever the signs of old endocardial mischief consist solely of a systolic murmur heard at the apex, and no first sound is heard either at the apex or in the axilla, or at the angle of the left scapular, referred pain is absent as a marked feature of the case.

I carefully exclude from this statement all cases where a systolic murmur is heard owing to the failure of a heart that has hypertrophied from other causes than endocarditis (*e.g.*, chronic Bright's disease). I also exclude those cases of acute rheumatism in which a systolic murmur appears during the attack. Table III., p. 186, shows a series of cases in which these conditions were fulfilled. They exhibited neither referred pain, referred headache, nor superficial tenderness of the chest or scalp throughout a period of observation extending from a minimum of six weeks up to a year.

Such cases, however, suffer from palpitation on exertion, and this palpitation is accompanied by shortness of breath and occasionally by a pain which is said to be situated over the apex of the heart. This pain does not go through to the back or round the body like a referred pain, and is unaccompanied by headache or by superficial tenderness of the trunk or scalp. It is apparently a true local pain.

(b) The second group of cases consists of those in which a mitral diastolic murmur is audible together with a systolic murmur. The systolic murmur is widely heard and conducted to the angle of the left scapula. No first sound is audible either at the apex or in the axilla, or at the angle of the left scapula.

Such physical signs are not associated with referred pain connected with the heart. As, however, the later stages of mitral stenosis fall into this group, referred pain may be

TABLE III.—MITRAL SYSTOLIC MURMURS AT THE APEX, OBSCURING

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.		
				1ST.	2ND.	SYSTOLIC.
25	F. 17	$\frac{5}{\text{Just Extl. N.L.}}$	Systolic	O	O	Heard widely over back
26	F. 14	$\frac{5}{1 \text{ Extl. N.L.}}$	O	O	X	Conducted to angle of scap.
27	F. 12	$\frac{5}{\frac{3}{4} \text{ Extl. N.L.}}$	Systolic	O	X	Heard widely over back
28	F. 21	$\frac{5}{\text{In N.L.}}$	O	O	X	Heard widely over back
29	F. 23	$\frac{5}{\frac{1}{2} \text{ Extl. N.L.}}$	O	O	2	Heard widely over back
30	M. 16	$\frac{5}{\text{In N.L.}}$	Systolic	O	2	Conducted to angle of scap.

TABLE IV.—MITRAL DIASTOLIC ACCOMPANIED BY SYSTOLIC MURMUR AT

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.		
				1ST.	2ND.	SYSTOLIC.
31	M. 23	$\frac{5}{\text{In N.L.}}$	Diastolic	O	2	Conducted to angle of scap.
32	F. 19	$\frac{5}{\text{In N.L.}}$	O	O	X	Conducted to angle of scap.
33	M. 42	$\frac{6}{1 \text{ Extl. N.L.}}$	O	O	2	Conducted to angle of scap.
34	M. 18	$\frac{5}{1 \text{ Extl. N.L.}}$	Diastolic	O	2	Conducted to angle of scap.
35	F. 17	$\frac{6}{1 \text{ Extl. N.L.}}$	Systolic	O	O	Conducted to angle of scap.
36	F. 18	$\frac{6}{\frac{3}{4} \text{ Extl. N.L.}}$	O	O	O	Conducted to angle of scap.

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pulse—"Virtual Tension"]. On the other hand, *low tension* means that the pulse is com-
 In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated;
 In the column devoted to the 1st sound at the apex, + means that this sound is

THE FIRST SOUND AT THE APEX, AND IN THE LEFT AXILLA.

DIAST.	PRESYS-TOLIC.	PULM. 2ND SOUND	PULSE.	VEINS OF NECK.	LIVER	LUNGS.	REFERRED PAIN AND TENDERNESS.
O	O	+	100 reg. low tension	O	O	Crackles at bases	No pain. No tenderness.
O	O	+	84 reg. low tension	Slight pulsation	O	R. base crackles	No pain. No tenderness.
O	O	+	120 reg. low tension	Slight pulsation	O	Crackles at bases	No pain. No tenderness.
O	O	+	116 reg. low tension	Slight pulsation	O	O	No pain. No tenderness.
O	O	+	84 reg. low tension	O	O	O	No pain. No tenderness.
O	O	2+	66 reg. low tension	Slight pulsation	O	O	No pain. No tenderness.

THE APEX, OBSCURING THE FIRST SOUND AT THE APEX AND IN THE AXILLA.

DIAST.	PRESYS-TOLIC.	PULM. 2ND SOUND	PULSE.	VEINS OF NECK.	LIVER	LUNGS.	REFERRED PAIN AND TENDERNESS.
X	O	+	74 irreg. maintained	O	O	O	No pain. No tenderness.
X	O	+	120 reg.	O	O	O	No pain. No tenderness.
X	O	+	60 irreg.	O	O	Crackles both bases	No pain. No tenderness.
X	O	+	88 reg. maintained	O	O	L. base	No pain. No tenderness.
X	O	+	76 reg.	O	O	Crackles L. base	No pain. No tenderness.
X	O	2	52 irreg.	O	O	O	No pain. No tenderness.

stroke is small, the tension, such as it is, is maintained between the beats [Broadbent's pressible, and whatever the initial stroke the tension is low between the beats. 2+ means that the 2nd sound is re-duplicated, but one element is accentuated. short, sharp, and high-pitched, as is typically found in marked mitral stenosis.

TABLE V.—SYSTOLIC MURMURS HEARD AT THE APEX

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.		
				1ST.	2ND.	SYSTOLIC.
37	M. 21	$\frac{5}{\text{Intl. N.L.}}$	O	+	X	Not conducted
38	F. 36	$\frac{5}{\text{Extl. N.L.}}$	O	+	X	Not conducted
39	F. 22	$\frac{5}{\frac{1}{2} \text{ Extl. N.L.}}$	Presystolic	+	X	Conducted
40	F. 29	$\frac{5}{\text{Intl. N.L.}}$	Diastolic	+	X	Not conducted
41	F. 33	$\frac{5}{\text{Intl. N.L.}}$	O	+	X	Not conducted
42	F. 17	$\frac{5}{1 \text{ Extl. N.L.}}$	Diastolic	+	2	Conducted
43	M. 42	$\frac{5}{\text{Intl. N.L.}}$	O	+	X	Not conducted
44	F. 38	$\frac{5}{\text{Intl. N.L.}}$	Systolic	+	X	Not conducted
45	F. 42	$\frac{5}{\text{In N.L.}}$	Systolic	+	X	Not conducted
46	F. 36	$\frac{5}{1 \text{ Extl. N.L.}}$	Diastolic	+	?	Not conducted

TABLE VI.—MITRAL DIASTOLIC OR PRESYSTOLIC MURMUR. No

No.	SEX & AGE.	APEX BEAT.	APICAL THRILL.	APEX.		
				1ST.	2ND.	SYSTOLIC.
47	F. 24	$\frac{5}{\text{N.L.}}$	O	+	X	O
48	M. 27	$\frac{5}{\text{N.L.}}$	O	+	X	O
49	M. 30	$\frac{5}{\text{N.L.}}$	Diastolic	+	X	O
50	M. 30	$\frac{5}{\text{Intl. N.L.}}$	O	+	X	O
51	M. 37	$\frac{5}{\text{Intl. N.L.}}$	Presystolic	+	X	O
52	F. 49	$\frac{5}{\text{N.L.}}$	Presystolic	+	X	O
53	F. 50	$\frac{5}{\text{N.L.}}$	O	+	X	O

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pulse—"Virtual Tension". On the other hand, *low tension* means that the pulse is com-
 In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated;
 In the column devoted to the 1st sound at the apex, + means that this sound is
Post Mortem Examinations. Case No. 43.—Mitral valve much stenosed [diameter
 Mitral valve much stenosed; converted into flattened funnel [diameter with cone

TOGETHER WITH A SHORT, SHARP, FIRST SOUND.

DIAST.	PRESYS-TOLIC.	PULM. 2ND SOUND	PULSE.	VEINS OF NECK.	LIVER	LUNGS.	REFERRED PAIN AND TENDERNESS.
X	?	+	76 reg. maintained	O	O	O	Stomach, back, tmpls. L. D ₆ & D ₇
O	O	+	100 reg. maintained	Pulsation	O	O	Stomach & back. D ₅ to D ₈ L. & R.
O	X	+	100 reg. maintained	O	O	Crackles both bases	Stomach & back. L. D ₆ and D ₇ .
X	X	+	64 reg. maintained	O	O	Crackles both bases	Stomach & back. D ₆ and D ₇ R. & L.
O	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D ₅ to D ₈ .
X	O	+	88 irreg. maintained	Pulsation	O	Crackles both bases	L. breast & back. L. D ₆ .
	O	+	88 irreg. maintained	O	O	Crackles both bases	Stomach & back. L. D ₆ and D ₇ .
O	O	+	100 reg. maintained	O	O	Crackles both bases	L. breast & back. L. D ₅ , D ₆ , D ₇ .
X	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D ₆ .
X	X	+	80 irreg. maintained	Slight pulsation	O	Crackles both bases	Stomach & back. L. D ₅ , D ₆ D ₇ .

MITRAL SYSTOLIC. FIRST SOUND SHORT AND SHARP AT APEX.

DIAST.	PRESYS-TOLIC.	PULM. 2ND SOUND	PULSE.	VEINS OF NECK.	LIVER	LUNGS.	REFERRED PAIN AND TENDERNESS.
O	X	+	70 reg. maintained	O	O	O	On exertion under L. breast & back.
X	X	2+	60 irreg. maintained	O	O	O	On exertion under L. breast & back.
X	X	+	54 reg. maintained	O	O	O	On exertion only.
O	X	+	80 irreg. maintained	O	O	O	On exertion.
O	X	+	80 irreg. maintained	O	O	Crackles at bases	O
O	X	+	84 irreg. maintained	O	O	O	O
X	X	+	60 irreg. maintained	O	O	O	O

stroke is small, the tension, such as it is, is maintained between the beats [Broadbent's pressible, and whatever the initial stroke the tension is low between the beats. 2+ means that the 2nd sound is re-duplicated, but one element is accentuated. short, sharp, and high-pitched, as is typically found in marked mitral stenosis. measured with cone 0·8 in.]. Left ventricle somewhat hypertrophied. Case No. 51.— 0·58 in.]. Left ventricle not hypertrophied.

present from enlargement of the liver, or affection of other organs than the heart. I have therefore not included such cases of cardiac failure in the short tabular arrangement (Table IV.) that I have drawn up to illustrate this statement.

(c) When a mitral systolic murmur is heard together with a sharp first sound at the apex or in the axilla, referred pain tends to appear as a symptom of the disease. Such a systolic murmur is usually not conducted into the axilla or heard at the angle of the left scapula, but this rule is frequently broken. It is, however, rarely if ever heard all over the back, and when heard at the angle of the left scapula is not infrequently accompanied by the first sound. This class of case may occasionally be characterised only by the above-mentioned signs, but usually a presystolic or diastolic murmur, and occasionally both murmurs are heard in addition to the systolic apical murmur. [Table V.]

By the statement that referred pain tends to appear in this class of case, I do not mean that it is continuously present. Thus, when the patient is at rest it is usually absent; but any undue exertion or any deterioration in the general condition will be signalled by the appearance of referred pain and headache. Moreover, a complaint of more or less pain is a feature in the history of such cases if they are watched over a considerable period of time.

(d.) A fourth group of cases of mitral disease consists of those in which no systolic mitral murmur is present. Either a presystolic, a diastolic, or both murmurs may be present, and the first sound has the typical short, sharp character usual in stenosis. The second sound is usually heard at the apex, and such cases thus belong to what Broadbent calls the first stage of mitral stenosis. [Table VI.]

In cases of this kind referred pain is not usually present. It may make its appearance as the sequel of some unusual act of exertion, or on first getting up after a considerable rest in bed; but nevertheless, referred pain is the exception rather than the rule during the time these signs are actually present.

I have so far spoken of referred pain in disease of the mitral valve without mentioning its position or characteristics. In such lesions as cause pain, it is situated within an area bounded above by a horizontal line at the level of the nipples and below by the line from the umbilicus to the costal border, and thence straight to the centre of the back. Not that the pain occupies the whole of this area at any time. It is mostly situated in the fifth space below the heart, at the epigastrium, and in the back just below or just internal to the angle of the scapula. It is situated mainly on the left side of the chest and back, sometimes entirely on the left side. The pain is not usually sharp and darting, but is, as a rule, dull and aching in character. It never becomes paroxysmal, or shows a tendency to spread suddenly and widely like the pain of aortic disease. It disables the patient less, but at the same time is more constant than the pain of aortic disease.

This pain is accompanied by superficial tenderness of greater or less extent over the sixth, seventh, and eighth dorsal segmental areas. The ninth dorsal also appears to be occasionally affected, but never without one or more of the above-mentioned areas. I therefore feel inclined to think that the implication of the ninth dorsal is due to spread downwards.

This tenderness is always worse on the left side, and the tenderness on the left side is the first to appear and the last to disappear.

The headache is situated in the temples and on the vertex, and more or less superficial tenderness is usually present over the temporal, vertical, parietal, segmental areas of the scalp.

Case 43, to illustrate referred pain produced by lesions of the mitral valve.—Peter H., aged 42. In 1882 he had rheumatic fever in which his feet and ankles were swollen and painful.

In 1892, one evening, after coming from work he spat a little blood. A month later he again spat a little blood after coming from work. He had suffered from pain in the chest for some time, and at last was compelled to go to a doctor (at the end of 1892), who said his heart was affected. He has been under the

doctor on and off ever since. He drank somewhat heavily till 1886, when he married; since then has been sober.

December 19, 1894.—Medium-sized man of 42; looks about his age. Face dusky, with a blue-red flush on both cheeks. No cyanosis of extremities, no œdema, no jaundice, no obvious dyspnoea. Says he becomes short of breath if he exerts himself much. Lies on his right side in bed. He complains of pain on

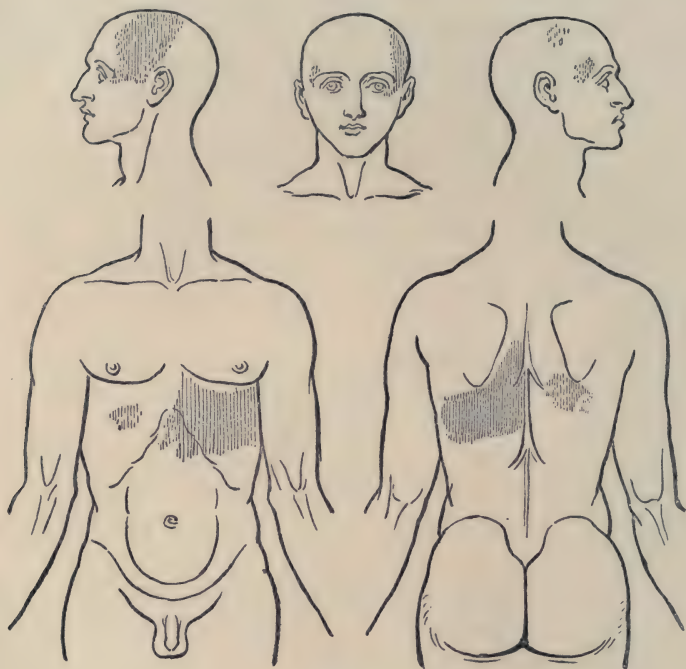


FIG. 5.

To show the extent of the superficial tenderness in a Case of Mitral Stenosis [Case No. 43], characterised by the following signs—a sharp first sound at the apex, followed by a systolic murmur not conducted to the angle of the left scapula.

The areas mainly affected on the trunk are the 6th and 7th dorsal on the left side. On the scalp the temporal and vertical areas are affected, mainly on the left side.

the left side of the chest in the fifth and sixth interspaces, and in the back close to the angle of the scapula. There is distinct superficial tenderness over both points.

Pulse 80, irregular both in rate and force. The irregularity in force is more marked than the irregularity in rhythm. Arterial wall not hard.

No pulsation of veins in the neck. Heart's apex beat seen and felt in fifth space $4\frac{1}{2}$ inches from the middle line (11.5 cm.), just outside the left nipple line. Impulse weak and irregular; no thrill.

Cardiac dulness begins above at the third space, extends about $4\frac{1}{2}$ inches (11.5 cm.) to the left and to the mid-sternum on the right.

Over the apex, the first sound is distinct, but sharp and thin; but further to the left in the axilla the first sound is distinctly sharp and high-pitched. The second sound is repudiated. A systolic murmur is audible over the fifth and sixth spaces from the left edge of the sternum to the line of the anterior axillary fold. Its maximum intensity lies over the apex beat, and no murmur is audible in the mid-axilla or at the angle of the left scapula. *Over the left second and third spaces* the second sound is accentuated; *over the right second and third spaces* the first sound is weak, the second sound somewhat slapping.

Respiration 24, regular. Alæ nasi and accessory muscles of inspiration are not working. No cough heard; no expectoration.

There are no abnormal signs in the lungs beyond those of slight general emphysema and a few moist crackling sounds at both bases.

Tongue clean, not furred. Appetite good, no vomiting; no pyrosis or eructation. Bowels opened daily. Urine 1020 acid. No albumen.

January 3.—He complained of pain in the epigastrium and below the angle of the left scapula. Marked band of superficial tenderness marking out the seventh dorsal area. Headache over the left temple with superficial tenderness over the left temporal area. Pulse 88, very irregular. Tongue clear, no vomiting. Physical signs as below.

January 4.—Exactly the same pains, headache and tenderness.

January 5.—The pain and headache are more marked, but are still practically confined to the left side.

The superficial tenderness both of the trunk and scarp are now very marked (fig. 5), and slight spots of tenderness have begun to appear on the right side. Tongue clean, moist. The pain is somewhat increased after taking food, but it is always there whether he takes food or not, and all food of every kind increases it. No vomiting; bowels still opened daily.

Pulse very irregular and of small stroke. No pulsation of veins in neck. Cardiac impulse felt diffusely in fifth space with its maximum above in his nipple line. Some epigastric pulsation. Suspicion of an occasional diastolic thirst.

Cardiac dulness beginning above in third space, extends about four inches to the left and about half-inch to the right of middle line of chest.

Over apex, first sound accentuated and sharp, second feeble. Systolic murmur not conducted into axilla or heard at angle of

left scapula. *Over left second and third spaces* second sound less markedly accentuated than before; *over right second and third spaces* first sound heard, second weak. Signs in lungs as before.

January 9.—The pain and tenderness became more accentuated, but occupied the same area as in fig. 5, except that the tenderness is more marked over the right side than in that figure.

January 15.—He continued in this condition till the evening of January 14, when the liver enlarged and signs of fluid appeared in the abdomen. To-day the pain is more marked on the right side than the left. He points to the right half of the abdomen from the costal margin to the iliac fossa and says he has pain in the right loins. The superficial tenderness extends from roughly the level of the seventh rib on the right side down to a point drawn from about the fourth lumbar spine forwards to a point about three inches below the umbilicus (seventh, eighth, ninth and tenth dorsal segmental areas). On his left side more or less tenderness is present over the same areas, but it is less well marked.

He now has tenderness of the scalp on both sides over the temporal, vertical, parietal areas, and over the right occipital area. This scalp tenderness is somewhat more marked on the right side than on the left.

Cardiac impulse very feeble. Cardiac dulness now extends $2\frac{1}{2}$ inches (6.5 cm.) to the right of the middle line of the chest and $4\frac{1}{2}$ inches to the left.

Over the apex, first sound weak, second reduplicated. No murmur of any kind can be heard. *Over pulmonary area*, second sound is scarcely heard. *Over aortic area*, sounds are extremely feeble.

Pulse about 76, very irregular, and with very small beat. Pulsation in the veins of the neck. Liver distinctly felt below the right costal margin. Signs of some fluid in the abdomen. Considerable quantity of æreated mucous expectoration, some of which is, however, browner than the rest. Cannot lie down in bed. Some crackling râles at both bases. No dulness.

Died January 17, 1895. The *post-mortem* revealed the following condition in the heart:—Right auricle somewhat dilated; right ventricle considerably dilated; left auricle much dilated, wall tough and fibrous; muscular substance diminished, excepting round the base of the appendix where it is hypertrophied; wall of left ventricle hypertrophied, but not greatly so; tricuspid valve dilated, smooth, no vegetations. From the auricular aspect the mitral valve appears as a long buttonhole slit with the edges in apposition throughout. It is very rigid. Fine bead-like vegetations on the free surface of the valve. The diameter of the valve measured with the cone is 0.8 inches (2 cm.). From the ventricular aspect the valve appears as a thickened, continuous ring of smaller diameter than from the auricular aspect. No marked change in aortic or pulmonary semi-lunar valves.

No micro-organisms discovered in the vegetations on the valve. Lungs congested. No infarcts, no consolidation. Liver, 58 ozs.; typical nutmeg.

Now the position of this referred pain and tenderness corresponds with that in affections of the stomach. We are thus brought face to face with the difficulty that the same areas may be affected in painful states due to lesions of the mitral valve and disturbances of the stomach. It might quite justly be urged that the presence of this pain and tenderness was due solely to a concomitant gastric disturbance; for troubles of digestion are not infrequent in these cases, and lend support to this objection.

I am unable to definitely prove my contention that this pain and tenderness is referred from the heart or disprove the objection that they are due to the stomach, but I think that a consideration of the following points tends to show that, in the majority of these cases, this pain and tenderness is of cardiac, rather than of gastric origin.

(1) True gastric disturbances are more common when the tricuspid valve fails and stagnation takes place in the system of the inferior vena cava. But it is exactly such a condition in which the left-sided pain and tenderness I have described is absent.

(2) In most cases where the first sound is abolished by a systolic murmur that is widely conducted referred pain is absent. But such cases not infrequently exhibit troubles of digestion.

Moreover, if the left ventricle fails, the first sound which was previously short and sharp may disappear, and a widespread systolic murmur be heard; at the same time, if a presystolic murmur was previously audible this murmur disappears. Under such conditions referred pain and tenderness may disappear to reappear again when the first sound returns under treatment. To illustrate this point I give the following case in which I twice watched this disappearance and reappearance of the first sound at the apex:—

Case 54.—To illustrate the disappearance of the referred pain, with alteration of the physical signs, and its reappearance when the physical signs improved again.—Rachel R. (V.P.H., Dr. Heron), aged 38, housewife.

First attack of St. Vitus's dance at the age of 13, second attack at 17. In 1875, at the age of 19, had an attack of acute rheumatism.

Since that time her heart used to beat quickly when she hurried, but she did her housework, and bore seven living children without complication.

1890.—She got a bad cough, and became very short of breath. Admitted to V.P.H. (Dr. Smith); went out well.

1892.—She became pregnant, and at the end of the fifth month came into V.P.H. again, with failure of the heart. Child born August, 1893, and three weeks afterwards again came into the hospital. From this time my observations on this case begin. She had evidently suffered for years from an ordinary type of mitral stenosis, and the least diminution in her general health would cause the left ventricle to fail.

September 4, 1894.—She had been admitted three weeks before with a systolic murmur at the apex, abolishing the first sound; the apical second sound was weak and reduplicated, and the pulmonary second sound was accentuated and single. There was no oedema of any part. She recovered in her usual manner.

August 30.—Menstruation began, and was accompanied by a good deal of pain in the lower abdomen and back. September 2. She was allowed up for half the day.

To-day there is a very marked change in her condition. She is taking no digitalis, so that this source of error is avoided, but menstruation still continues. Ears and lips are somewhat blue. No jaundice. No oedema anywhere. She has no pain, no headache, and no superficial tendency anywhere, but she complains that she feels faint and sick. She lies propped up in bed, but there are no marked objective signs of dyspnoea. Pulse small, and very irregular. Very slight pulsation of the veins in the neck. Apex beat in sixth space, five inches (12.5 cm) from mid-sternum. No thrill. Epigastric pulsation. Cardiac dulness begins above at the third rib, extends one and a half inches (4 cm.) to the right, and five and a half inches (14 cm.) to the left of the mid-sternum.

Over the apex, a loud systolic murmur is heard conducted into the axilla, but not heard at the angle of the scapula. Outside the nipple line and in the axilla the first sound is very feeble, but as we pass inwards there is a small area in the fourth and fifth spaces, extending for about one and a half inches (4 cm.) to the left of the left sternal edge, over which the first sound is short and sharp. The second sound is reduplicated. *Over the left second and third spaces* the second sound is accentuated, and not reduplicated. *Over the lower right sternal border* first sound is fairly well heard, and second sound is accentuated and single. Beyond a few crackling sounds at both bases, which were always audible in this case, there were no abnormal signs in the lungs. Tongue clean and moist. Nausea, but no vomiting. Complete loss of appetite. Bowels opened. Liver not enlarged. Urine

thirty ounces in twenty-four hours; no albumen. She was kept strictly to bed, and given tinct. strophanthus $\mathfrak{m}\mathfrak{v}$., with extr. convallaria gr. v., every six hours. Menstruation ceased on September 9.

September 18.—She has greatly improved. The feeling of faintness, sinking, and giddiness is passing away. She has begun to take food well. Pulse now 88, irregular in force and rate, but more so in force than rate. Tension now maintained. Apex beat four inches from middle line, and cardiac dulness extends one and a quarter inches to the right and four and a quarter inches (11.25 cm.) to the left of the middle line of the sternum.

Over lower part of left back and in left axilla a loud, sharp, accentuated first sound is audible, but no murmur. *Over the apex* the first sound is still audible, but is obscured by a short systolic murmur. At the line of the anterior axillary fold both the sharp first sound, so well heard in the axilla, and the systolic, heard over the apex, are audible together. Second sound over the apex reduplicated. *Over the left second and third spaces* second sound accentuated and single.

By September 15 the pulse was nearly regular, and a typical short, sharp, accentuated first sound was now heard over the whole area from the left sternal border to the angle of the left scapula behind. Over the apex beat it was accompanied by a short systolic murmur. No murmurs within the ventricular diastole heard anywhere.

September 18.—All the faint, sinking feeling, the feeling of sickness, and the shortness of breath have been absent for some time, and have not reappeared. To-day pain has begun to appear again, and she is complaining of pain over the lower ribs under both breasts and under the scapula, more marked on the left side than the right. There is definite superficial tenderness over portions of the seventh and eighth dorsal areas on both sides. Tongue clean. Appetite good. Bowels opened daily. Pulse 90, somewhat irregular in rate. Tension maintained. The cardiac signs are as on September 13, excepting that a præ-systolic murmur of short duration now precedes the short, sharp, accentuated first sound. The systolic murmur is heard over the front of the chest only, and is not conducted into the axilla or to the angle of the left scapula. At the angle of the scapula the first sound is distinctly heard. From this point onwards a præ-systolic or diastolic murmur was audible, in addition to the local systolic murmur.

September 22.—She went home, engaged in house work, &c., and on September 27 came back in the same condition as on September 4. No sign of a præ-systolic murmur; the apical first sound is entirely abolished at apex, in axilla, and at angle of left scapula. All the sinking faintness had come back, and she had orthopnœa as before; but all pain and superficial tenderness were absent.

(3) The referred pain and tenderness I have described may make their appearance quite apart from food, when the patient first gets up after a long stay in bed, or after some special exertion. This is best seen in those cases where pain and tenderness are not a marked feature. Case No. 47 showed this point well.

(4) If the pain is present it may be aggravated by food, but it may entirely disappear by rest in bed, or by other treatment directed towards the heart, although the diet remains quite unchanged.

(5) I think that the pain over the shoulder joint and in the left side of the neck (accompanied by superficial tenderness over the fourth cervical segmental area) which in gastric disturbances is so commonly associated with pain and tenderness within the areas we are considering is, as a rule, absent in lesions of the mitral valve. Thus, although the pain under the heart at the epigastrium, and in the region of the shoulder-blade is shared by both disease of the stomach and certain lesions of the mitral valve, the pain and tenderness over the shoulder-joint is not common to these two diseases.

The value of these considerations can only be gauged by repeated observations of cases of mitral disease; but, so far as my experience goes at present, I cannot help believing that this referred pain is due, under the conditions I have described, to the cardiac condition, and not to the stomach.

§ 6. *Referred Pain due to Enlargement of the Liver produced by Failure of the Right Side of the Heart.*

As a rule, no referred pain can be definitely associated with the right side of the heart. For tricuspid regurgitation is apparently unaccompanied by referred pain and tricuspid stenosis is of such rarity that no one physician can obtain sufficient cases for observation.

But when the tricuspid valve yields, an exceedingly important focus of referred pain not uncommonly makes its appearance in consequence of the distension of the liver. All cases do not show this referred pain, but it most often appears when the liver enlarges for the first time, especially if this enlargement takes place with considerable rapidity.

If the liver remains enlarged the pain may entirely disappear in a few days, and if we have reason to think that the liver is hard and fibrotic, or, if enlargement has frequently occurred before, it may be completely painless.

When the liver suddenly enlarges for the first time, the patient complains of pain over the right hypochondrium, iliac fossa, and in his right loin.

If pressure is made over the enlarged liver the patient complains of pain, and the liver is therefore said to be tender. Now it is quite probable that the liver itself is really tender, but in the large number of cases the supposed tenderness of the liver is found to be in reality superficial. Thus the tenderness is well marked in the right loin and when tested in the usual way the superficial structures of the abdominal wall and back are found to be tender. If well developed, the eighth, ninth, and tenth dorsal segmental areas can be marked out. The important fact in such cases is the presence of the tenth dorsal area. For the presence of the eight and ninth dorsal areas on the right side may be due to the occurrence of doubling from the left side, and may be simply a sign that the pain and tenderness from some cardiac lesion on the left side has become very intense. But no cardiac lesion causes primary tenderness between the level of the umbilical line, and, therefore, the presence of the tenth dorsal area which lies below this line, points in cardiac cases directly to the liver.

The same area is also usually present on the left side of the abdomen and back, but the tenderness is less intense, more spotty in distribution, appears later, and disappears earlier than that on the right side.

With the appearance of this tenth dorsal area on the body tenderness usually appears over the occipital region of the scalp. But as the eighth and ninth dorsal areas are also present on the right side of the scalp we usually find that the headaches and tenderness of the scalp are distributed over the vertical parietal and occipital areas.

An exceeding good instance of the referred pain and superficial tenderness due to secondary enlargement of the liver is given in fig. 6.

The tenderness is, however, usually somewhat wider spread when the liver enlarges.

Case 55.—Sarah S. (Victoria Park, Dr. Eustace Smith), aged 25. Single.

July 1893.—Acute rheumatism. Remained in bed a month and when she got up found that her breath was very short.

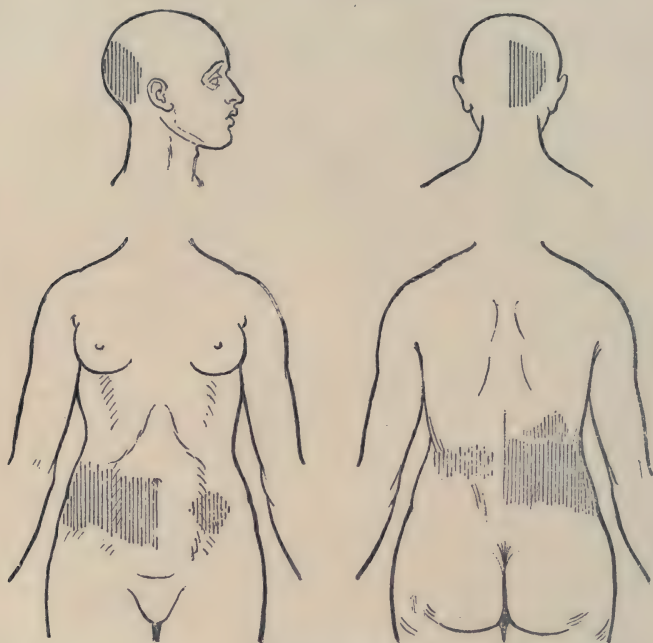


FIG. 6.

To illustrate the pain and tenderness over the 10th dorsal area, produced by Acute Enlargement of the Liver, in consequence of Cardiac Failure [Case No. 55].

Note the tenderness over the occipital region of the scalp.

September, 1893.—Admitted to V.P. Hospital with mitral stenosis and regurgitation.

Apex beat diffuse and forcible. Diastolic apical thrill. Upper limit, cardiac dulness third rib. Right limit, right edge of sternum. Left limit, nipple line. At apex first sound is loud and followed by a systolic murmur conducted as far as the left axilla. Second sound heard at the apex and followed by a short diastolic murmur. The pulmonary second sound was reduplicated. Aortic second sound normal.

Pulse small, irregular, easily compressible.

Edge of liver just felt.

She much improved, and was discharged on December 13, 1893.

As soon as she was discharged she tried to walk about the house, within a few days began to vomit. Much pain in the abdomen and headache.

Readmitted December 29, 1893. Condition as follows:—

Skin of face of a slightly yellowish tinge with a red spot on each cheek. Conjunctivæ not yellow. Ears and lips a little blue. No swelling of ankles. Distinct clubbing of the fingers. Knee-jerks normal. Plantar reflexes brisk.

She cannot lie flat in bed. Very short of breath on the least exertion. No fainting, but frequently feels faint.

She complains of pain in the abdomen below the navel and in the loins; she also has pain "across the shoulders and over the thorax below the level of the nipples." Much superficial tenderness.

Headache over the occipital region and over both temples associated with superficial tenderness of the scalp.

Pulse about 84, irregular both in force and rhythm, small stroke, compressible.

Distinct pulsation, but no marked distension of the veins of the neck.

Apex beat in fifth space in the nipple line. Pulsation also seen in third and fourth spaces internal to nipple line. Upper limit of cardiac dulness third rib. Right limit, cardiac dulness right edge of sternum. Left limit, cardiac dulness outside the nipple line. Heart's action very irregular. With the short beats a systolic murmur only is heard, but when the diastola is prolonged a soft diastolic murmur is heard. The first sound is feeble at the apex, but can be heard independently of the systolic murmur. The systolic murmur is conducted to the angle of the left scapula. No murmur heard over aortic area. First sound feeble over the right edge of the sternum.

Liver a good 2 inches (5 cm.) below the border of the ribs.

Suspicion of fluid in the abdomen.

Respiration 22. Slight cough; worse at night.

Some crackling sounds heard at the bases of both lungs and at apex of right lung.

Tongue red and a little furred. Bowels opened daily. She frequently vomited undigested food.

She has not menstruated for five months. Usually lasts two days. No pain.

January 9.—The feet are swollen to-day and there are distinct signs of fluid in the abdomen.

The liver edge can now be felt just above the level of the umbilicus.

She complains of pain in the abdomen below the umbilicus and occipital headache.

Marked superficial tenderness of the abdominal wall over the area in fig. 6, with definite tenderness of the occipital region of the scalp.

The liver continued to enlarge and the pulse became very irregular and small. She died on January 20, 1894.

On January 6 the temperature began to rise and oscillated irregularly between 100° and 98° F. (37·8° and 36·7° C.)

The urine was acid, sp. gr. 1025, and contained an amount of albumen which varied from about $\frac{1}{12}$ to $\frac{1}{10}$.

Post-mortem. — No pericarditis. Right side of the heart dilated. Tricuspid orifice admitted four fingers. Pulmonary valves otherwise normal. Left ventricle hypertrophied. Some thickening of the edges of the mitral valve which was somewhat stenosed. The valve was incompetent. Around its base on the auricular surface was a small layer of bead-like vegetations. The aortic valves showed fresh vegetations, which contained streptococci in considerable numbers.

No peritonitis. Fluid in the abdomen.

Liver weighed three pounds six ounces. Congested and fibrotic.

Kidneys congested, but otherwise normal.

Spleen small and congested with small fresh infarct in upper part.

Left lung healthy except for congestion at the base. Right lung congested. In middle lobe was a small infarct with some collapse around it.

Pelvic organs normal. Ovaries showed no signs of recent mēnstruation.

§ 7. *Paroxysmal Pain of Cardiac Origin.*

Writers on cardiac diseases express the most diverse opinions in their attempts to define the disease “angina pectoris,” but are practically unanimous with regard to the symptoms of an “anginal” attack. Given the symptoms in any one case, the one group of observers refuse to diagnose angina pectoris if the physical signs of cardiac disease are present; whilst, at the opposite pole, are those who speak of angina pectoris whenever the symptoms are present, regardless of the age of the patient or the physical signs of disease in the heart.

But if differences of opinion are found between the various authorities during the life of the patient, still greater differences manifest themselves at the autopsy. Some cling to one anatomical lesion, some to another, and every case

that does not show a particular lesion is spoken of as false, or pseudo-angina pectoris.

From the point of view of the nomenclature of disease it is of obvious utility to confine the name *angina pectoris* to cases of a certain clinical type (*e.g.*, the classical cases in elderly men), or, to those which show some simple anatomical lesion (*e.g.*, occlusion of the coronary arteries.)

But throughout this study I am not engaged in a description of diseases, but in a physiological investigation of pain by the help of cases of disease. In this attempt to unravel the origin of pain it is necessary to look to physiological causes, and not to those descriptive characteristics which would enable a naturalist to separate one species from another. For the same anatomical lesion may produce different physiological effects, and the same physiological effects depend upon many different anatomical lesions. Thus, for my purpose, it would be a grave mistake to confine this section to a study of only one clinical class of paroxysmal cardiac pain. And yet I should be the last to attempt to bring all the cases here considered under the heading of a single disease.

In those cases, therefore, in which I am obliged to call the case by the name of some disease, I shall speak of primary *angina pectoris* where the symptoms are present without gross physical signs within the heart, and secondary *angina pectoris* where physical signs exist pointing to lesions of the aorta, or its valves.

Now, the symptoms of an anginal attack may be grouped under three main headings.

(1) A feeling of suffocation, accompanied by a pain at the epigastrium. This pain is variously described as "pressing pain," "bursting pain," "like being tied in with a cord," "as if the heart was too big."

(2) A sense of impending death. The face is said to become cold, the jaws stiff, and then this cold, "deathly" feeling spreads over the body.

(3) Violent radiating pains mostly over the upper part of the chest, and in one or both arms. These are accompanied by marked and wide-spread superficial tenderness, and are thus truly referred.

In any individual case these three symptoms may be present to a very varying degree. Thus my first illustration (Case 56) is chosen because of the complete development of these three groups of symptoms in the attacks. Yet for the purposes of classification of disease this man was probably a case of angina pectoris secondary to aneurism of the aorta.

Case 56.—To illustrate the complete phenomena of an anginal attack in a man with aneurism of the aorta. William D. (V.P.H. Dr. Eustace Smith), aged 38, railway porter.

At the end of 1892 he was out of sorts, and found he could not lift such heavy weights as before. One morning in January, 1893, he had a feeling of suffocation and giddiness, and his heart began to beat very fast. He felt faint and had to sit down. The doctor said he had "valvular disease." Throughout the next six months he had frequent attacks, in which he had a sudden feeling of suffocation with a choking sensation in the throat, and accompanied by a pain in the epigastrium as if a cord was pressing it in. In June, 1894, his attacks altered in character, and became like those seen in the hospital. These attacks came on particularly during his work.

He drank heavily till three years ago, and has been occupied with exceedingly heavy lifting work as porter at a goods' station. Denies syphilis.

On admission (October 1, 1894). Largely built man of 38. Face of a uniform pinkish-yellow colour. No anæmia. No cyanosis. No jaundice. No œdema. Exceedingly well nourished. Looks as if he had gained weight.

Pulse 90, regular, somewhat high tension. Arterial wall a little hard for a man of 38. Pulses equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat very feeble, and cannot be definitely localised. No pulsations over the upper part of the chest. No thrill.

Cardiac dulness begins at third rib, extends $\frac{3}{4}$ in. (2 cm.) to the right of the middle line of the sternum and $5\frac{1}{2}$ in. (14 cm.) to the left of the mid-sternal line.

Over the upper part of the sternum, and in the second right interspace, a sharp and ringing second sound is heard. The first sound is inaudible; and a distinct systolic murmur is heard over the upper part of the sternum to the aortic area. It is not conducted over the apex; the first sound is very short and wanting in tone, and closely resembles the second sound, so that the sounds at the apex have a "tick tack" character closely resembling that of a

fœtal heart. No murmur heard at the apex, or at the angle of the left scapula.

No cough or expectoration. He is short of breath on exertion, and cannot sleep flat upon the back.

No abnormal physical signs in the lungs.

Tongue moist and a little grey. Appetite poor. No vomiting. Bowels confined. Does not usually suffer from flatus.

Liver and spleen not free.

The pupils are equal to react to light and accommodation. The left does not dilate quite so well to shade as the right.

Urine normal.

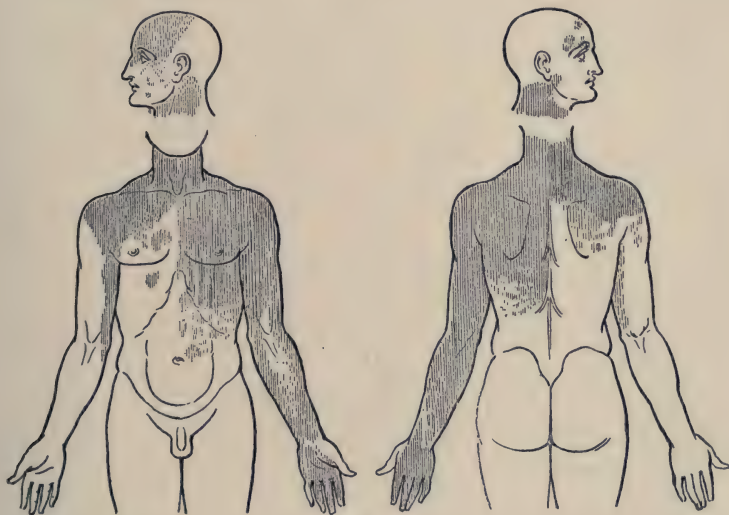


FIG. 7.

To show the widespread tenderness present after a major anginal attack in Case 56.

Nature of the Attacks and of the Pain.—On October 7 he was completely free from pain. During the night, at 1 a.m., on October 8, a patient in the ward became violent. William D. was sitting up to watch the struggle when he was noticed to lie back with his head thrown back over the pile of pillows, and his neck extended. His face was sweating, and he said he had a cold, dead feeling over the forehead, nose, and lips, which spread all over his face. The jaws felt stiff. He felt exactly as if he were dying. As he lay back he had a feeling over the region of the apex beat, and in the epigastrium, as if something were getting tighter and tighter. Then a shooting, tearing pain came on over the second and third interspaces, rapidly followed by a piercing pain over the anterior and posterior aspects of the left shoulder joint. Then the pain ran

over the left sterno-mastoid to the occipital region, and down the left arm to the top of the little finger. At the same time he suffered from marked headache over the left half of the forehead. During the later part of the attack, his heart was beating rapidly, but was not irregular. The attack lasted two minutes. It was followed by considerable mental depression, and he said he felt utterly exhausted. He complained of widespread soreness, and said he could scarcely bear his shirt, owing to the tenderness of his chest and back. (Fig. 7.) About a quarter of an hour after the attack he passed a considerable quantity of pale urine.

This represents a complete, fully-developed attack, characterised by the following symptoms:—(1) feeling of coldness; (2)



FIG. 8.

To show the tenderness present after an attack of paroxysmal pain [minor attack of the first type].

feeling of death and suffocation; (3) local bursting feeling at the heart; (4) violent shooting referred pains in chest and head, followed by intense widespread superficial tenderness. But during a long period of observation it became plain that he also suffered from minor attacks of two separate types.

First type (October 21, 1894). He had not slept well on account of pain in the upper part of left side of chest and back. In the morning his chest ached and felt sore. At 10 a.m. the pain began to shoot, and to spread as in a major attack, but it was of much less intensity. This shooting gradually ceased, and left behind it a sore aching feeling wider spread and more intense than before. Superficial tenderness was present on the left side of the

trunk, from the level of the jaw to the horizontal level of the tenth dorsal spine. (Fig. 8.) It extended down the ulnar border of the arm to the little finger. Some tenderness, but to a much less degree, was present on the right side of the chest. The whole fronto-temporal, mid-orbital, and frontal and nasal areas of the scalp and face were tender. There was no feeling of death or suffocation, and no sensation in the throat. This type of attack occurred when he was recovering under rest in bed and treatment with nitro-glycerine.

Second type (March 12, 1896). He woke feeling quite well. Whilst washing himself he raised both hands to the top of his head, and immediately sank back on to his pillows with a feeling of tightness across the chest. He felt suffocated, as if he would never get his breath again, with a cramped feeling in the throat as if something was being tightened. He felt a rolling, fluttering, irregular movement at the heart. It passed off, but returned again in a few minutes, and for an hour and a half he continued to have repeated small attacks of this nature. Before they began he was completely free from pain, but throughout the hour and a half during which they lasted a feeling of soreness over the upper part of the left chest and in the left arm gradually developed. There was no shooting or stabbing pain, as in the major attack, but a slow increase, with each little attack, of the sore aching pain. Finally he showed exceedingly marked superficial tenderness of the left side of the chest and back, from the level of the jaw to the horizontal level of about the tenth spine, extending down the left arm. There was very slight tenderness on the right side of the chest. Marked tenderness of the anterior part of the scalp. This type of attack seemed to occur when he returned to work after the major attacks had been abolished for a period of many months by treatment.

March, 1896.—The physical signs are still exactly as on admission in October, 1894, except that the systolic murmur is somewhat less marked.

Now this man not only had many major attacks, demonstrating all the symptoms of angina pectoris, but he also had minor attacks of two distinct types of much importance in attempting to analyse the phenomena of a major attack.

The one type was always preceded by some referred pain in the arm and shoulder. The attack consisted in the rapid spread of this pain to other parts. The heart did not become irregular. The sense of impending death was absent.

The second type came on after a definite exertion, and consisted of small exacerbations in a condition which was

present for an hour or more. He felt a tightness across the chest, a cramped feeling in the throat, and a rolling, irregular feeling at the heart. The heart seems to beat irregularly. These sensations lasted almost two minutes, leaving him with an uncomfortable feeling as if he would drop, and then recurred again and again at short intervals. On one occasion the whole state lasted one hour and a half. When these attacks begin there is no pain, but referred pain slowly appears, and becomes more marked after each little attack of suffocation. Finally, at the end of a considerable period, the pain and superficial tenderness become more marked. Thus the main feature of this type of attack is the suffocative feeling, and referred pain is not developed until these attacks are repeated many times.

Now just as this case showed minor attacks of two separate types, so we find that cases of primary and secondary angina pectoris range themselves at a point somewhere between two extreme types. On the one extreme lies the type in which the feeling of suffocation or strangulation and the sense of impending dissolution predominate. Referred pain may actually be absent, or if it is present, it simply follows the cardiac disturbance exactly as we should expect. On the other extreme lies the type that usually suffers from more or less pain and superficial tenderness apart from the attack. Here the spread of the referred pain and its increase in intensity are the main features of the attack. The heart's action is at first undisturbed, but as the pain continues to spread the heart begins to beat, not only with great rapidity but with increased force. The sense of suffocation and impending death may be absent.

Case 57 more nearly approached the first extreme type. He was an old man with profoundly degenerate arteries. The sounds of the heart were exceedingly feeble, and a musical systolic murmur was audible, apparently originating at the aortic valves. The attacks occurred in groups, and consisted firstly of palpitation and a sense that he was dying, and then secondarily of pain. The superficial tenderness before the first recognised attack was slight, but after the attack was over it had spread widely.

Case 57.—To illustrate the type of anginal attacks, in which the suffocation and sense of impending death are the main features, and referred pain and tenderness a secondary phenomenon.—John K. (V.P.H., Dr. Eustace Smith), aged 64; a canvasser.

In 1888 he began to suffer from palpitation of the heart. In 1889 he was in the hospital; but at that time suffered from no paroxysmal attacks. In 1890 he began to have slight attacks in which he felt faint. They began with gasping; he then felt as if he was going to die. Pain was not a marked feature of these attacks. In November, 1893, pain became a marked feature of the attacks, which then assumed the form described below.

He drank heavily as a young man, but has been a teetotaler for ten years. Has had many accidents, but no diseases, excepting one attack of gout in the left toe.

On admission. Medium-sized man of 64; looks considerably older. Marked arcus senilis. No cyanosis. No jaundice. No cedema. No wasting.

He has never fainted. On exertion he suffers from palpitation, which he says is quite different from the palpitation of the attacks. Not very short of breath, excepting during the attacks. Sleeps with two pillows, but does not need to be otherwise propped up.

Pulse 72, regular; of increased tension. The arterial wall is excessively tortuous and rough to the feel. Every artery of any size is tortuous and visible. The pulses are equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat seen and felt in fifth space $\frac{1}{2}$ inch (1.5 cm.) outside nipple line. Not heaving. No thrill. No other pulsation visible in chest.

Cardiac dulness much diminished by emphysema. All over the front of the chest, within the areas enclosed by the right and the left nipple line is heard a high pitched, exceedingly musical systolic murmur. It is not conducted to the angle of the left scapula. Over the aortic area both first and second sounds are inaudible, whilst over the apex beat the first sound is not heard, but the second is occasionally very feebly audible. The weakness of the sounds of the heart was a very noticeable feature of this case.

Respiration 20: with the expiratory period prolonged. Marked elevation of the chest. Alæ nasi quiet. Single, non-paroxysmal cough; not very troublesome now. Very small quantity of grey mucous expectoration.

Physical signs in chest those of emphysema, but none of bronchitis.

Tongue clean, moist. No vomiting.

Liver and spleen not enlarged.

Urine 1010, acid. No albumen or sugar.

Nature of the pain and of the paroxysmal attacks.—The following account of an attack seen on February 24 is typical

of all his present attacks. In the morning he complained that he had slept badly, and felt a good deal of soreness in his chest. (Fig. 9). There were several spots of superficial tenderness confined to the left side of chest and inner aspect of left elbow. He had no headache, but there was some superficial tenderness above and around the left eye. At 10.30 p.m. he suddenly sat straight up in bed and said he was going to have an attack. His pulse was beating at the rate of 150 in the minute. He was sweating over the face, but there was no marked alteration of colour. Thirty seconds after the beginning of the attack he began to wheeze violently. In sixty seconds the pulse dropped in rate, and became very irregular. He complained that his heart felt as if

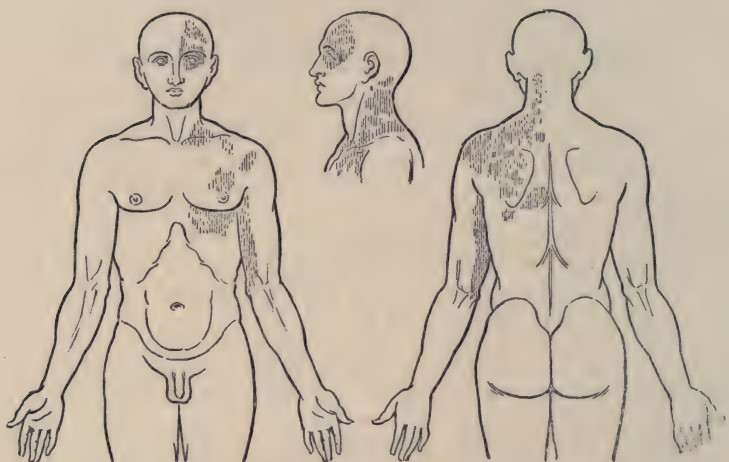


FIG. 9.

To show the tenderness which was present before the attack of angina pectoris in Case No. 57.

The dotted portions are those over which the tenderness was ill-defined and of no marked intensity.

it were swelled. The attack was over in two and a half minutes. He said that, when he called me, the palpitation had begun at the heart. Then the pain began to gather force. He had had pain in the chest and left arm before, and these pains rapidly increased, and were joined by a pain in the region of the hyoid. He complained that the skin of his forehead felt bulged, and that his nose felt swollen. Profound and widespread superficial tenderness of scalp, chest, and arms after the attack.

At 11 p.m., whilst I had my hand upon his pulse, he had a second attack. In the first ten seconds the heart beat 27 (at the rate of 162 in the minute). At the beginning of the second half minute it beat 24 in ten seconds (144 in the minute). At the

beginning of the third half minute it beat 14 in ten seconds (84 in the minute), but was very irregular both in rhythm and force. The pulse at the wrist was now very feeble and irregular. At the beginning of the fourth half minute it still beat 14 in ten seconds. At the end of the first half minute he began to wheeze violently and to clutch at his throat, complaining that he suffered pain there. The pain in the chest became much worse, and he felt as if he were dying. His body and head were sweating, and his hair became quite moist. He then gave a great yawn, and the attack was over.

He always insisted that the increase in the shoulder pain did not begin till after his attack was well on him; and that the attacks began with palpitation and were followed by pain.

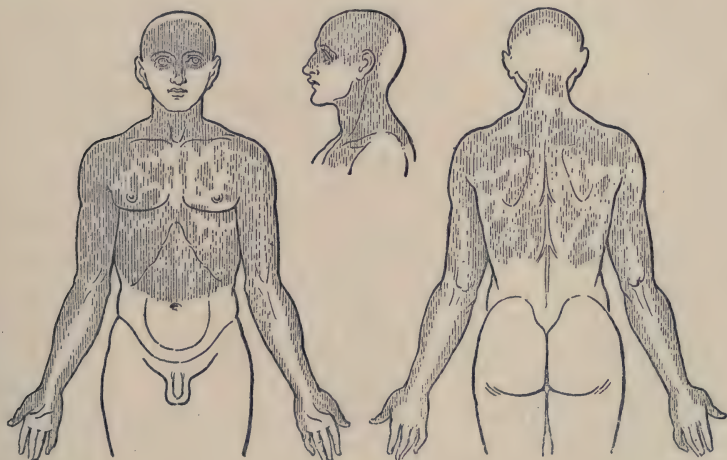


FIG. 10.

To show the extent of the superficial tenderness after the attack of angina pectoris in Case 57.

After the attack he had much widespread feeling of soreness of trunk and arms, and a feeling as if his head had swelled and tightened his scalp. Much widespread superficial tenderness of body and scalp. (Fig. 10.) The left arm felt numb, as if he had been lying on it. He could make no fine movements of his fingers, but there was no absolute paralysis. The loss of power seemed to him to be below the elbow. He seemed to be utterly exhausted, and was very depressed after the attack was over. Shortly afterwards he passed a considerable quantity of pale urine.

During the attack the systolic murmur was still heard, but neither first or second sound were audible anywhere. There were no moist sounds in the lung during the stage of wheezing.

These attacks enormously improved under treatment, and particularly with strict rest in bed. During the time he was in bed he had a few slight attacks, and it was very evident that the referred pain and superficial tenderness followed the attacks and were directly produced by them. For he would have an attack when all referred pain and superficial tenderness were absent, but after the attack marked referred pain and superficial tenderness would make their appearance mainly over the left side.

On the other hand, Case 58 more nearly approached the second extreme type. This case suffered from aortic regurgitation and stenosis as the sequel of acute rheumatism, and similar phenomena are found in some cases where the aortic regurgitation is of non-rheumatic origin. Here the attacks consisted mainly of a violent radiation of the referred pain present before the attack. An appreciable time elapsed between the beginning of the radiation of pain and the increase in the rapidity and force of the heart's beat.

It is interesting to notice that when the left ventricle failed after a series of attacks and a mitral regurgitant murmur became audible, abolishing the apical first sound, all attacks ceased. When the heart recovered the attacks returned. It is also interesting to note that this case died, not in an attack, but from cardiac failure.

Case 58.—To illustrate an anginal attack in which the spread of referred pain was the primary phenomena. Mary S. (V. P. H., Dr. Sainsbury), aged 18. In December, 1888, she had her first attack of rheumatic fever, which left her with an affection of the aortic valves. She remained fairly well, excepting that she suffered from pain in the chest and shortness of breath, until June, 1889, when she had a second attack of acute rheumatism. When she recovered from this second attack she had pain in the chest more or less continuously. Shortly afterwards, at 9 p.m. one night, she had her first attack.

From July, 1893, to March, 1894, she was under my observation day by day, and the following account is a *resumé* of the notes taken during this period by myself.

She is a finely-built rather stout girl of 18; looks a good deal older. Face pale, with occasional irregular flushes on cheeks and chin. No œdema; no jaundice; no cyanosis; no fainting; short of breath on exertion, and she occasionally suffered from periods during which she could only breathe when propped up. Palpitation on exertion and occasionally when lying in bed.

This palpitation always leaves her with a pain in the left breast and left temple, and if pain was present before, it is markedly increased by the palpitation.

Pulse, as a rule, about 80, regular, but easily runs up on exertion; typically low tension, with a strong stroke and marked regurgitant characteristics. Arterial pulsation visible in neck as high as the ears.

Extreme capillary pulsation; no venous pulsation. Cardiac pulsation visible over whole left half of chest. What appears to be the apex beat can be felt in the fifth space in the line of the anterior axillary fold. Cardiac dulness begins at second space above, extends to the right edge of the sternum and to a point to the left on the fifth rib in the line of the anterior axillary fold.

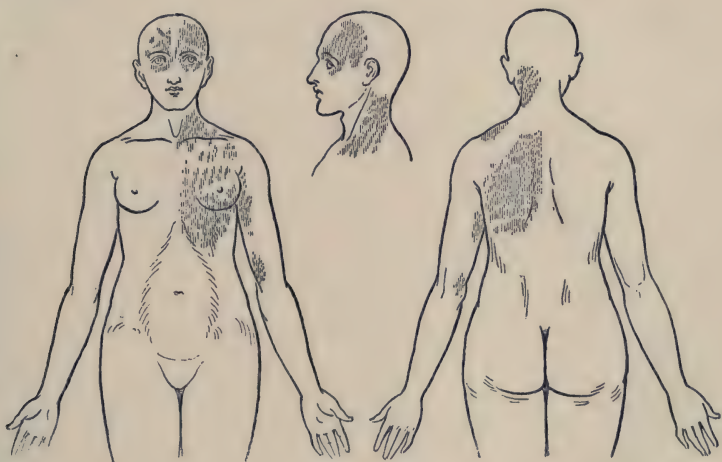


FIG. 11.

To show the extent of the superficial tenderness in Case No. 58, apart from an attack of angina pectoris.

Over the aortic area neither first nor second sound are heard but all over the sternum is a loud double aortic murmur (systolic and diastolic). Over the apex beat both first and second sounds are audible. The first sound is also heard at the angle of the left scapula. There is no mitral murmur either at the apex or angle of left scapula.

No abnormal physical signs in the lungs. Tongue clean; appetite bad. No pain after food; no vomiting; bowels somewhat confined. Liver and spleen not enlarged. Urine 1030 acid, no albumen, no sugar.

Nature and distribution of the pain and tenderness.—She usually complained of pain over the left breast and in the left interscapula region of the back. At times the pain was situated

over the inner side of the left arm. More or less superficial tenderness was usually present, usually on the left side, but sometimes, if the pain was severe, on both sides of the chest. The most common distribution of this tenderness was between the second and fifth rib in front, and from the level of the third dorsal to the seventh dorsal spine behind, and the tenderness involved either the upper third or the whole of the inner aspect of the arm to the elbow. (Fig. 11.) It was always most marked over the left half of the chest and back, and remained longer on the left side than the right.

The headache extended from the fronto-temporal region forwards over the eyes to a greater or less extent. This headache was accompanied by superficial tenderness of the forehead, and

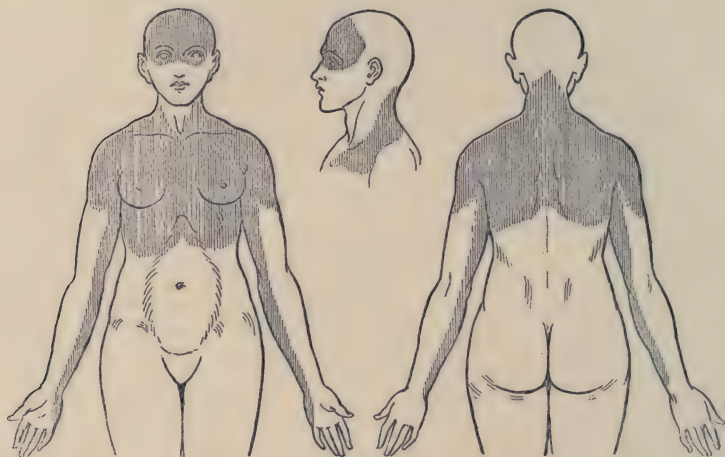


FIG. 12.

To show the extent of the superficial tenderness in case No. 58 during the time the pain was spreading, and 3 minutes before the quickening of the heart, in the attack of angina pectoris described in the text.

anterior portion of the temporal regions; it was usually bi-lateral, but occasionally unilateral confined to the left side. (Fig. 11.)

The attacks, which occurred at one time as often as once in twenty-four hours, were as follows: she suffered from pain for hours or even days before an attack. As a paroxysm came on, she would call out that she was going to have an attack. She said she felt the pain spreading. (Fig. 12.) She said it was going down the left arm and into the shoulder. The forehead was moist, the lips of a grey white, the area around the mouth pale, but the face still retained its pink colour. The pulse was 80. Then she called out that the pain was all over her chest. She threw herself backwards, arched her back, and clung to the rail at the head of the

bed. The forehead and cheeks flushed, and intense capillary pulsation was visible all over the face; the arteries of her neck pulsated intensely. The pulse ran up to 160, and became of extremely low tension. The heart beat seemed to shake the whole front of the chest. The arching of the back then ceased, the arms fell flat on the bed and she began to cough. The pulse rate rapidly fell. The attack occasionally ended in crying, and after it was over she generally passed a considerable quantity of pale urine of low specific gravity. It was followed by the most intense and wide-spread superficial tenderness of the thorax, back, arms, and scalp. The extent of the tenderness over the scalp and trunk closely resembled that shown on Fig. 10. The whole attack lasted about three minutes, but occasionally the first attack had scarcely passed off before a second came on.

As the pain spread it was noticed that the superficial tenderness became of much wider distribution. Thus I was, on more than one occasion, able to watch it spread down the inner side of the arm to the elbow, and then to the ulnar border of the hand. I also watched it spring into the neck and to the opposite side of the body. All this occurred before the pulse altered in rate. (Fig. 12.)

This I took to be an indication for treatment, and on several occasions, after the pain and tenderness had begun to spread, an injection of morphia or gr. xxx. of phenacetin prevented the great rise in pulse rate. The attack then completely altered in character. For the pain began to spread as usual, reached a certain point, accompanied by wide-spread superficial tenderness, and then slowly died away without the occurrence of the flushing, rapid pulse beat, or arching of the back.

During the attack she felt very faint, but the faintness was not sufficient to alarm her. She had no fear of impending death, although on more than one occasion the pulse became imperceptible at the wrist, and she seemed on the point of death.

The permanent pain, and the frequency of the attacks, were much lessened by treatment, and she left the hospital. I saw her three months later in a condition, shortly, as follows: the double aortic murmur was present as before, but the first sound at the apex was replaced by a systolic murmur, which was audible at the left angle of the scapula; the liver was distinctly enlarged; the mitral valve had evidently failed. She had had no attacks for three weeks, and she remained free from attacks for a further two weeks until the mitral murmur disappeared. With the marked improvement in the general condition of the left ventricle the anginal attacks returned.

She died in December, 1894, of cardiac failure.

To understand how severe attacks, like those of Case No. 58, may be developed in cases of aortic regurgitation, I give Case No. 59. Here the pain was, as a rule, not

paroxysmal, but closely resembled that usually seen in aortic disease, when unaccompanied by mitral regurgitation. However, under the influence of slight exertion, distinct paroxysms of slight intensity, but of the same nature as those of Case No. 58, appeared at infrequent intervals. Such cases are not uncommon; and are very instructive for the study of the origin of paroxysmal attacks arising in patients with organic disease of the heart.

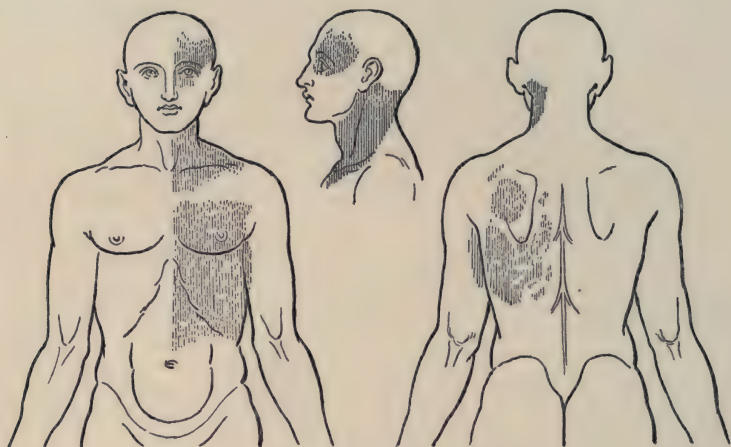


FIG. 13.

To show the extent of the superficial tenderness in Case No. 59, after the attack of "faintness" described in the text. Before the attack the tenderness was much less extensive.

Case 59.—To illustrate the origin of paroxysmal attacks of pain in cases of organic disease of the heart. Samuel F. (V.P.H. Dr. Eustace Smith), aged 40; labourer.

About Christmas, 1891, he was running to catch a train when he felt a pain "at the heart." This pain was exceedingly sharp, and forced him to stop. It never quite left him; and in April, 1893, it began to affect his left arm, but he continued with his work in spite of it until November, 1893. He was then seized with great shortness of breath, and suffered from much pain. A bad cough came on, and he spat up blood-stained phlegm. He was admitted to the hospital on December 11, 1893.

No history of acute rheumatism could be obtained, and he denies all venereal disease. His work is exceedingly heavy; and consists in unloading chests of oranges from the hold of a vessel,

and carrying them on his back up a flight of stairs into the warehouse.

On admission.—He is a well-built man of 40, and looks quite his age. No anæmia. No jaundice. No œdema. No clubbing of the fingers.

He fainted several times before admission, and within the first twenty-four hours after admission, felt faint more than once. He says his heart beats irregularly when he exerts himself. He is very short of breath when he walks about, but not when he lies in bed. He lies on the right side in bed on account of the pain in the left side.

He complained of pain over the inner side of the left arm as far as the elbows. Occasionally the pain runs down to the fingers of his left hand, and he has a stinging, numbing feeling in the middle, ring, and little fingers of the left hand. He also complained of pain at the epigastrium, and over points above and internal to the nipple, and under the outer part of the clavicle. All the pain was on the left side.

He also complained of headache in the left temple, extending over the left half of the forehead.

There was marked superficial tenderness over the chest, neck, temple, and forehead on the left side only.

Apex beat is feeble, but is felt in fifth space as far out as 2 in. (5 cm.) outside the left nipple line. No pulsation felt elsewhere in the chest.

The cardiac dulness begins above, at the third space, extends to the right sternal border, and to rather over 1 in. (2.5 cm.) outside the left nipple line.

Over the whole of the sternum, and over the apex, a loud aortic diastolic murmur is heard. No systolic murmur is audible. At the aortic cartilage the first and second sounds are not heard, whilst both sounds are distinct at the apex.

Pulse 100, regular, somewhat low tension. Pulses are equal on the two sides.

No cough or expectoration now.

No abnormal signs beyond a few *râles* at the right base.

Tongue a little furred. No vomiting now. No pain after food.

Liver and spleen not enlarged.

December 31.—He was free from pain until he got up to tea. At teatime he complained of a pain which started under the left breast and in the epigastrium. This pain then spread to the upper spaces, and then travelled slowly until it involved the left half of the neck and the inner side of the left arm. He then had a feeling as if he was going to drop. When I reached him he had crawled into bed. He was crying, and said he felt faint. The aching pain was still severe, but had ceased to spread. He was sweating profusely.

He showed exceedingly marked tenderness of the superficial structures of the chest, back, and neck, as in fig. 13. Headache

and tenderness were also present over the left temple and forehead.

There was no marked alteration in the physical signs, but the pulse was somewhat more rapid. The temperature had risen to 100° F. (37·8° C.)

He remained in bed for three weeks, and was treated with a pill containing extr. belladonna gr. $\frac{1}{4}$, pulv. opii gr. i. Although he had some occasional pain he had no further attacks.

Thus we must conclude that paroxysmal cardiac pain can be developed in one of two ways:—

(1) The pain is the expression of the cardiac disturbance and consists of a local epigastric pain, followed by more or less widespread referred pain. The attack is accompanied by a feeling of impending death and suffocation.

Attacks, more or less approaching this type, are best seen in men well on in life with calcareous arteries and feeble heart sounds.

(2) The pain is the main feature. The attack consists in the rapid radiation and increase in intensity of the referred pain that has been present before the paroxysm began. The feeling of suffocation or impending death may be absent.

Such attacks require a visceral lesion to produce the antecedent referred pain. They are, therefore, best seen in cases of aortic regurgitation, whether of rheumatic origin or not.

CHAPTER II.

THEORETICAL.

§ 1. *The Conditions within the Heart which lead to Referred Pain.*

UP to this point I have only attempted to show an association between certain physical signs and the presence or absence of referred pain. I shall now consider the significance of these signs, and attempt to show why they are associated with referred pain.

In an ordinary case there can be but little doubt about the presence of aortic stenosis and regurgitation. However, it

is not always so easy to be certain of the amount of the stenosis or the regurgitation. For it is only necessary to remind the reader that a systolic murmur over the aortic area, with the distribution of a murmur of aortic stenosis, may appear as a consequence of a roughening of the valves only, without any true stenosis. In the same way, a diastolic murmur, resembling the murmur of aortic regurgitation, may be present without abolishing the aortic second sound. In both these cases the pulse will help to show that but little stenosis is present in the one case, and little regurgitation in the other.

True aortic stenosis throws more work on the wall of the left ventricle during systole. The ventricle, therefore, hypertrophies, compensation takes place, and the patient suffers from no symptoms. Not so aortic regurgitation. For here the ventricle empties itself during systole, but as soon as the wall relaxes, at the beginning of the diastole, the blood pours back from the aorta and distends its cavity. In pure aortic stenosis the ventricle has to perform increased work during the systole, but is at rest during diastole. In pure regurgitation, on the other hand, there is not necessarily any increase in the work required during systole; but just at the moment when the wall is not toned to meet an effort, the blood comes pouring back into its cavity from the aorta. Now it is this distension, at the moment when the walls of the cavity are at rest, that is most pernicious, and it is this condition, above all others, that is most liable to lead to referred pain.

When both regurgitation and stenosis are present the condition is still worse. For not only has the systole to overcome a greater intra-ventricular resistance, but the diastole is broken by a back rush of blood from the aorta. The left ventricle hypertrophies to meet this increased resistance in front, but the increase in the force of the ventricular contraction will also increase the force with which the blood rushes back from the aorta at the beginning of the diastole. Thus referred pain is more likely to be present with aortic regurgitation than with stenosis, but most certainly when both lesions are combined.

But pressure within the cavity of the ventricle during the diastolic period gives rise to a second evil. The walls of the cavity are particularly liable to dilate, and the contents of the cavity to permanently increase. The ventricular wall now acts at a disadvantage on its contents, and they may possibly not be completely expelled during the systole. Thus the ventricle is never actually empty, but is always somewhat distended, an ideal condition for the production of referred pain. In most cases this is probably but a temporary condition, arising from some additional exertion in a heart normally working at the top of its power. So long as the patient remained more or less at rest, the ventricle is able to empty itself during systole; but it is working at the extreme limit of its power, and a little extra difficulty will render the complete emptying of the cavity at each systole impossible. In young people some excessive effort is necessary to produce this condition, but in older people, and especially in those cases where the lesion of the aortic valve is not of rheumatic origin, it may arise after any small exertion, such as getting up after a rest of a day or two in bed.

This permanent distension of the left ventricle cannot exist long, or to any very great extent, without the mitral valve becoming incompetent. Directly this happens the pressure within the ventricle becomes relieved, and the cavity is again emptied of blood at each systole. An enormous load is taken off the walls of the ventricle, for the blood can now leave its cavity by two roads. If the resistance in front is great, a considerable quantity of blood is now pumped, at each ventricular systole, into the pulmonary circulation, through the incompetent mitral valve, for the highest pressure in the lungs is low compared with that in the aorta. The aorta then receives less blood, the pressure within it falls, and the tension in front is relieved. Thus, as soon as the mitral valve yields, the permanent pressure within the left ventricle is relieved by a self-regulating mechanism, and referred pain disappears.

In the same way the essential conditions for the production of referred pain are absent in those cases where the

incompetence of the mitral valve arises coincidently with disease of the aortic valves during acute rheumatism.

Thus, whether the yielding of the mitral valve be a primary or a secondary occurrence, free regurgitation through this orifice acts as a safety vent to the ventricle, and prevents that distension of its cavity during diastole, necessary for the production of referred pain.

So far, the physical signs, upon which we have depended for a knowledge of the condition of the circulation in valvular disease, are fairly easy of interpretation. But a similar attempt to translate the physical signs produced by disease of the mitral valves, is beset with many difficulties. I will, therefore, begin with those cases where the interpretation of the physical signs presents least difficulty.

If a systolic murmur, audible at the apex and well conducted to the back, abolishes the first sound at the apex, in the axilla, and at the angle of the left scapula, and especially if the pulse is at the same time one of low tension, the lesion of the mitral valve is probably pure incompetence.

In such cases the blood is expelled by each contraction of the ventricle, not only into the aorta, but also into the auricle. A free path is open into the pulmonary circulation, and the pressure in the ventricle, therefore, always remains low. With each diastole the ventricle dilates, and the blood flows in again from the auricle at the pulmonary pressure. Thus the heart has to act more frequently to circulate the same amount of blood, and the pulse is increased in rate, but its tension is low. As extra work has to be done by the left ventricle, its wall hypertrophies, and complete compensation may occur. It is true the auricle receives blood from the left ventricle during the auricular diastole, but the blood does not enter at high pressure. Moreover, the left auricle can empty itself freely at each stroke through the free opening of the mitral valve, and thus the mean pressure within its cavity does not rise. Hence such cases of pure mitral regurgitation are the least serious of all cardiac lesions, and are unattended by referred pain.

Let us now consider the condition of the circulation in a pure case of mitral stenosis, characterised by a pre-systolic

murmur of the typical quality, terminated by a sharp first sound. It is almost universally acknowledged that this murmur is produced by a jet of blood forced through the narrowed mitral orifice by the sharp contraction of a well-acting and probably hypertrophied auricle. For it is the most capricious of all cardiac murmurs; it disappears as soon as the heart shows signs of failing, but reappears as the patient recovers. It is always absent some time before death. If absent when the patient is at rest, it reappears when, by any exertion, such as lifting the arms, extra work is thrown on the heart. The blood enters the auricle during the diastole. Then follows the auricular systole, which drives the blood forcibly through the narrowed orifice, producing the pre-systolic murmur. The ventricular systole follows immediately, and terminates abruptly the flow from the auricles. Then follows the auricular diastole, during which the cavity of the auricle can remain at rest, except for the somewhat high tension in the pulmonary circulation, which always follows a narrowing of the mitral valve.

Under such conditions the pressure in the auricle is high, but the contractile force of the auricle is increased to meet it; the diastole of the auricle is uninterrupted, and there is nothing to cause referred pain. It is, of course, possible that some exertion, which, though actually not great, is relatively considerable, owing to the diseased condition of the valves, may upset this circulation. Blood would then accumulate in the auricle, and it would remain permanently distended, and referred pain would result. This probably accounts for the occasional presence of referred pain in these pure cases of mitral stenosis. Pain is, however, the exception, and only occurs after some definite indiscretion, and not during the even tenour of a quiet life.

But imagine that just when the powerful auricular systole has expelled the blood through the narrowed mitral orifice, a strong jet of blood is forced back into its cavity, just as its walls are relaxing, we should then have exactly the condition which we found so fruitful a source of referred pain in aortic regurgitation, except that the auricle here

bears the brunt of the diastolic pressure instead of the ventricle.

This is the condition that I believe exists in those cases where an apical systolic murmur is present, accompanied by a short, sharp, accentuated first sound.

The actual mechanism of this short, sharp, first sound in mitral stenosis is still uncertain. But whether its origin be valvular or muscular, it seems to be associated with the contraction of a well-acting left ventricle on a light load. If the ventricle loses power, and dilates, this sharp first sound disappears. At the same time, the systolic murmur may become audible over the back and in the axilla. Thus, whatever the mechanism of this accentuated, sharp, first sound may be, it seems to be associated with a well-acting left ventricle and a stenosed mitral orifice.

It might be objected that in pure mitral regurgitation blood is also forced into the auricle at the moment of its diastole by the contraction of the left ventricle, and yet that this condition gives rise to no referred pain. But we must remember that in such cases the mitral orifice offers no obstruction. The contracting auricle can empty itself with ease into the ventricle, and the blood which regurgitates from the left ventricle passes easily into the pulmonary circulation. In mitral stenosis, on the other hand, before the left ventricle dilates and the heart fails, the tension in the pulmonary veins and left auricle are high, owing to the obstruction to the onward flow. This increase of tension is evident from the frequency of hæmophysis in cases of mitral stenosis. Out of twenty-five consecutive cases, eighteen suffered from hæmoptysis at one time or another. Thus even a slight increase in the auricular contents during its diastole, especially if it take place with rapidity and considerable force, must act injuriously.

The following considerations also show that the regurgitation in these cases of considerable mitral stenosis occurs at a considerable pressure. The pulse of mitral stenosis, at any rate in the earlier stages, is of well-maintained tension, though the stroke is poor. Now, before blood can leave the left ventricle, and pass into the aorta, the tension in the

aorta must be overcome. Thus the contents of the left ventricle must be under fair pressure. If regurgitation takes place from the left ventricle, this regurgitation must, then, also take place under fair pressure. On the other hand, the pulse of pure mitral regurgitation is distinctly one of low tension, and easily compressible. This points to the regurgitation through the mitral orifice also occurring at low pressure.

Thus, in conclusion, it would seem that the most potent conditions for the production of referred pain are: firstly, the maintenance of considerable tension within a cavity of the heart, accompanied by, secondly, a sudden accession of tension (owing to regurgitation) at the moment when the walls of that cavity are dilating after systole.

Throughout this paper I have omitted all consideration of complex combinations of valvular lesions. For, although such cases form interesting problems for solution, when the principles which govern the presence or absence of referred pain are once determined, the conditions of the circulation are too complicated to aid in the determination of such principles. Moreover, the various groups into which such cases fall, do not occur with sufficient frequency. I have also neglected an interesting group of cases where referred pain appears as the accompaniment of acute endocarditis, arising during the course of an attack of acute rheumatism.

§ 2. *The Sensory Supply of the Various Portions of the Heart.*

I have attempted to show, in the previous section, that the referred pain, which occurs in certain cases of mitral stenosis, is due to conditions of pressure within the left auricle. Now, on examining the records of such cases (p. 186 to 189), we find that the referred pain and superficial tenderness lie within the areas from the fifth to the ninth dorsal, but mainly within the sixth and seventh dorsal segments. Thus we may assume that if the explanation of the causes which give rise to referred pain in such cases is correct, the sensory impulses from the auricle pass into the

central nervous system at the level of the fifth, sixth, seventh, eighth, and possibly the ninth dorsal segments, but mainly at the level of the sixth and seventh dorsal.

In the same way the referred pain, in cases of aortic regurgitation, or of combined aortic regurgitation and stenosis, were attributed to abnormal pressure in the left ventricle. Now the pain and superficial tenderness in such cases lies mainly over the second, third, fourth and fifth dorsal, with occasional implication of the fourth cervical and sixth dorsal. Thus the impulses from the left ventricle probably pass into the cord at these levels.

A study of aneurisms of the aorta (p. 168) taught us that the referred pain and superficial tenderness they produce may be divided into three groups. Firstly, with aneurisms of the ascending arch, the pain and superficial tenderness lie over one or more of the following areas—first, second, third dorsal, third and fourth cervical. Secondly, if the transverse arch is implicated, the pain and tenderness may lie over the anterior surface of the neck, especially within the inferior laryngeal area. Thirdly, when we have reason to think that the aneurism has involved that part of the aorta which lies beyond the entrance of the ductus arteriosus, the pain and superficial tenderness may lie within the fifth, sixth and seventh dorsal segmental areas.

These results may be tabulated as follows :—

Sensory Supply of the Heart.

Transverse arch of aorta	...	Inferior laryngeal segment
Ascending arch of aorta	...	3rd and 4th cervical 1st, 2nd and 3rd dorsal [? 4th dorsal.]
Ventricle	2nd, 3rd, 4th and 5th dorsal [? 6th dorsal.]
Auricle	5th, 6th, 7th and 8th dorsal [? 9th dorsal.]

§ 3. *The Physiology of Cardiac Pain.*

Pain does not exist without a cause. It is simply the expression of the stimulation of sensory nerves within the heart, and this stimulation has a definite aim.

The first effect of the presence of pain will be to warn the patient against exertion. Under the influence of this warning the body is placed more at rest, and the heart will have a chance of recovering, in consequence of the diminished call made upon it. Pain, wherever it occurs in organic heart disease, is a warning and an intimation that rest is required.

But, probably, the stimulation of the sensory nerves, of which pain is the expression, performs a second function. We know that the depressor nerve conducts sensory impulses from the heart to the medulla, which cause dilatation of the visceral capillaries, and so lower the pressure in the distal portion of the vascular system. We also know that augmentor fibres pass from the dorsal cord to the heart, increasing not only the rate but the strength of its contractions. Now, these fibres have been found in the second, third, fourth, and fifth anterior roots, and it is quite possible that the sensory portion of the reflex arc is of much wider extent.

Thus it is possible that these pain impulses may be the expression of the afferent arc of one, or both, of these reflex mechanisms; at any rate, in those cases where the pain lies over the upper dorsal or third and fourth cervical areas.

It is, possible, therefore, that the implicated segments of the central nervous system are both acted on, and react upon the heart. This must be borne in mind during the study of paroxysmal attacks of referred pain. A very widespread disturbance of the heart, especially if it occur suddenly, will cause extremely widespread referred pain, owing to the intensity of the stimulus poured into the central nervous system. Before the disturbance in the heart's activity, no referred pain or tenderness will be present; but during and after the manifestation, both will be intense and widespread. This is the one extreme type of anginal attack. But, supposing pain impulses of considerable in-

tensity have been pouring into the segments of the central nervous system for a considerable time, the resistance within the nervous system may ultimately break down, and the impulses will spread up and down the cord. When the cord is widely implicated, especially if the impulses are of considerable intensity, the motor mechanism will become involved, and not only will the tension fall rapidly [depressor action], but the heart will beat with greater rapidity and force [augmentor action]. This is the second extreme type of anginal attack, in which the antecedent referred pain is the main feature, and the cardiac disturbance appears to be secondary to the spread of the pain.

But in order that the stimulation of this reflex arc may occur, the heart must not be worn out by disease of long existence. For, as I pointed out above, much referred pain may appear when the liver enlarges for the first time; but if it remains enlarged, or if the enlargement has occurred many times, the pain ceases to make its appearance, owing to the changes undergone by the liver substance. In the same way, so long as the condition of the heart remains fair, referred pain appears when the necessary conditions for its appearance are fulfilled, and the heart then reacts to the stimulus so produced. But, later on, towards the end of the case, the heart becomes worn out, and structural changes occur. Such hearts react neither to changes of internal pressure nor to drugs; and the patient passes away painlessly, and in spite of all our efforts.

§ 4. *The Distribution of Referred Pain and Tenderness in Diseases of the Heart considered from the Developmental Aspect.*

In section 2 of this chapter I attempted to map out the sensory supply of the heart, and the result must appear, at first sight, somewhat startling. For it would appear that the ascending arch of the aorta receives its innervation from the highest segments, whilst the impulses from the auricle enter the lowest segments that stand in relation to the heart. Thus the sensory visceral innervation of the heart is

upside down in relation to the present position of the adult human heart.

Now it is worth considering, shortly, what we know of the development of the heart in attempting to explain this remarkable fact.¹

Let us take up the development of the heart at the stage where the two tubes have fused to form a single tubular heart. The hinder end is continuous with the two vitelline veins, whilst the anterior end bifurcates into two primitive aortæ. At this time, the heart lies in the middle line under the upper part of the alimentary canal, and clearly shows the division into three portions. The hindmost of these becomes the auricle, the middle portion the ventricles, and the most headward portion the bulbus aortæ, and ultimately the ascending arch of the aorta. This tube then becomes bent on itself, so that the hindmost, or auricular portion, comes to lie above the middle or ventricular portion.

Now the plan upon which the sensory supply of the sympathetic system to the viscera is laid down, seems to be very antique, and to represent an early stage of vertebrate phylogeny. The sensory supply of the heart bears out this statement, in that the impulses enter the segments of the central nervous system just as if the auricles were still the hindmost portion of the heart, and as if the heart were a median organ.

That portion of the aorta, from the valves to the innominate artery (ascending arch) is innervated like the ventricle, from the second, third, (and, perhaps, the fourth) dorsal segments, and also refers into the first dorsal, and third and fourth cervical areas. Thus the next segment, headward of the first dorsal, is the fourth cervical. The fifth, sixth, seventh, and eighth cervical are skipped, and a gap exists, as I pointed out in my two previous papers.

¹The following considerations will be rendered clearer, to those of my readers who are not familiar with the development of the mammalian heart, by a reference to Figs. 176, 177, 178, 179, and 181 in Quain's "Anatomy," 10th edition, vol. i., part 1. The originals of these beautiful figures are found in His, "Anat. d. Mensch. Embryonen," a work that is in the hands of but few persons. I have, therefore, referred the reader to Prof. Schäfer's excellent modifications of the original figures.

Now the third and fourth cervical areas lie over a part of the neck, which is definitely somatic in origin, and has no connection with the structures formed from gill arches.¹ This area of skin grows forwards from the dorsal surface of the body, encroaching on the gill arches and their appendages. Now, as I pointed out before,² the sensory portion of the first cervical segment is contained in the trigeminal (fifth nerve), and the second cervical (whose sensory fibres enter in the cervical region at the proper level) receives its afferent impulses from the posterior part of the scalp. Thus the third cervical segment comes to be the highest segment which receives impulses from a true somatic skin surface, and is the uppermost segment which supplies true somatic viscera, *e.g.*, the ascending arch of the aorta, and the apex of the lung.

In front of the anterior border of each sterno-mastoid, lies a triangular area, which the dissector knows as the anterior triangle of the neck. Within this area referred pain and superficial tenderness appear, with lesions of such essentially branchial structures as the larynx and its appendages. The ventricles and the lung never primarily refer into this area. Thus, I pointed out that the areas within this region, like those of the jaws and face (excluding the nose, eyes, and forehead) belonged to a branchial system of segmentation, apart altogether from the somatic segmentation, and that of the areas on the forehead and scalp.

Let us turn for a moment to what we know of the development of the aorta in man and the higher vertebrates. The bulbus arteriosus is the most anterior or headward portion of the heart. It ends in front in a series of lateral vessels, which run from the ventral surface towards the back. The front four of these vessels run in the four branchial arches; of these the first three form the carotids and their branches, and do not concern us here. The fourth of these vessels, which runs in the fourth branchial bar, behind the

¹ Cf. Part II., BRAIN, 1894, p. 477; also His. "Anatomie Menschlicher Embryonen," Leipzig, 1885.

² BRAIN, 1894, p. 477.

third branchial cleft, forms the transverse arch of the aorta on the left side, and the innominate artery on the right side. The fifth vessel, which runs behind the fourth cleft, forms the pulmonary artery. On each side, dorsally, these five vessels are connected with one another. Thus, the fourth vessel, running in the fourth arch on the left side (the future aorta), unites, on the dorsal surface of the body, with the fifth vessel (the future pulmonary artery). The point at which this union takes place is marked on the adult aorta by the entry of the obliterated ductus arteriosus, which originally united the stem of the pulmonary artery with the aorta, and is the remains of the dorsal portion of the fifth branchial vessel. Thus we have now divided the aorta as we know it, in the adult with three portions: (1), from the semi-lunar valves to the level of the origin of the innominate. This is the representative in the adult of the bulbus arteriosus, the most headward division of the tubular heart. (2), from the innominate to the point of entry of the ductus arteriosus. This part represents, in the adult, the fourth branchial vessel. (3), all the aorta beyond this point—a true somatic dorsal vessel.

It is, therefore, peculiarly interesting to find that aneurisms of the ascending, or first part of the aorta, cause tenderness over the third and fourth cervical, and first, second, third dorsal segmental areas; all of which are purely somatic, and either identical with, or a continuation headwards of the areas to which the ventricle refers. On the other hand, aneurisms of the transverse arch, the remains of a true branchial vessel, cause pain and tenderness within the inferior laryngeal areas of the neck, in common with so truly a branchial organ as the larynx. This is the more instructive, from the fact that the larynx proper is developed from the structures of the fourth branchial bar, of which the transverse arch of the aorta represents the branchial vessel.

Much of what I have said in this section may be fanciful, and may be negatived by future investigation. But, at any rate, for the present, such embryological considerations seem to me to offer the only hypothesis to explain the distribution of referred pain and superficial tenderness in diseases of the heart and aorta.

CHAPTER III.

PAIN IN DISEASES OF THE RESPIRATORY ORGANS.

§ 1. *Character of the Pain.*

IN my first paper I scarcely touched on the pain produced by diseases of the lungs, and neglected the pain which may occur in the course of phthisis ; for whatever pain is present in this disease throws little light on the nature and causes of referred pain, and on the limits and anatomical distribution of areas of superficial tenderness. But although of but slight scientific interest for the determination of localisation, in the central nervous system, the distribution of pain and tenderness is of considerable practical importance, and occupies a considerable field in the clinical picture of many diseases of the lungs. The study of pain in its different forms in various diseases of the lung and pleura, also forms a useful demonstration of the methods by which widespread pain and large territories of superficial tenderness are to be interpreted.

Two quite distinct varieties of pain, accompanied by two even more distinct types of tenderness, may make their appearance in the course of diseases of the lung and pleura. Pain of the first type is local, and occupies no predetermined spots or areas. It is situated over the focus of disease, and usually in those intercostal spaces which cover a patch of pleural friction. It is accompanied by no true superficial tenderness, but marked deep tenderness is evoked by pressure or percussion. Pain of the second type is referred—that is to say, it is not necessarily situated over the focus of disease. It runs through or around the body, and has a focus behind and one in front. If severe, or of any duration, it is accompanied by more or less marked tenderness of the superficial structures of the chest, and this tenderness lies over predetermined spots or bands. Thus, referred pain and tenderness do not point directly, but only indirectly to the focus of disease.

(A) Local Pain in Pulmonary Diseases.

This type of pain occasionally appears in the neighbourhood of phthisical centres, or large areas of pneumonic consolidation; but in the large majority of cases it stands in definite relation to a pleuritic rub. It is said to be "stabbing" or "catching" in character. It is absent when the side is at rest, is intensified by each inspiration, and becomes agonising during the long and deep inspiration which follows a cough, a laugh, or a sneeze. It is relieved by any treatment that puts the side at complete rest, such as successful strapping. The patient describes the pain as like the sudden stab of a knife into the side. But this stab does not go through the chest from back to front, as is the case with severe referred pain, but is localised to one spot.

This pain is frequently associated with marked tenderness on pressure or percussion in the intercostal spaces.¹ Thus, by fairly heavy percussion, an area can generally be marked out within which deep tenderness exists. Now within this area pleuritic friction will usually be audible. I must warn the reader from supposing that referred pain and superficial tenderness never co-exist with an acute pleurisy. But where superficial tenderness, of the usual type that accompanies referred pain, exists, it is due to the disease of the lung, of which the pleurisy is a secondary phenomenon, and is not due directly to the pleurisy. For in a considerable number of cases of acute pleurisy all superficial tenderness is absent, and the presence of the patch of deep tenderness, corresponding more or less closely to the area of friction, can be easily demonstrated. Thus, before proceeding to test for deep tenderness, it is well to determine that all superficial tenderness is absent, or the results of the examination will be fallacious.

This deep tenderness, in most cases, seems to be definitely due to pressure upon the inflamed pleura. For it corresponds very fairly with the area of audible friction, and is less easily obtained over the ribs than over the inter-

¹ Walshe ("Diseases of Lungs," third edition, p. 269) recognised this tenderness on pressure.

costal spaces. It disappears with time, even though the friction may remain, and thus it is sometimes a useful guide to the duration of a friction sound, present when the patient first comes under observation. Local pain and deep tenderness usually disappear when serous fluid is effused; but I have more than once seen it persist over the upper part of the purulent effusion in empyema.

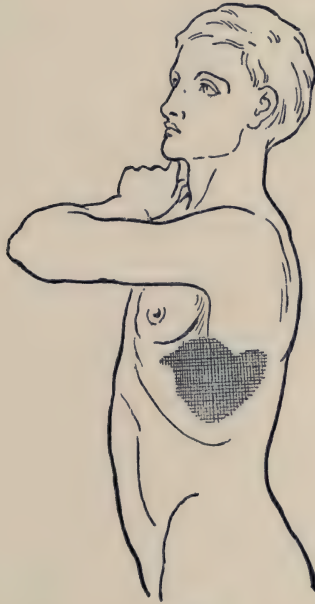


FIG. 14.

To show the area of deep tenderness evoked by pressure or percussion in a case of acute pleurisy [Case No. 60].

Case 60.—To illustrate the local pain and tenderness on deep pressure which accompanies a pleurisy. John K. (V.P.H., Dr. Heron); aged 44; coachbuilder.

In 1888 he is said to have suffered from pleurisy, but no detailed account of the attack can be obtained. Some cough each winter since then, but he has been able to do his work. Is a teetotaller. No family history of phthisis.

On admission (December 11, 1893).—Medium-sized well-built man of 44. Looks older. No marked anæmia. No wasting. No cyanosis. No night sweating.

Respiration, 24. No marked dyspnoea. Hard cough in paroxysms, which seem to choke him. Expectoration mainly aerated mucus. No tubercle bacilli found in sputum.

He complains of pain in seventh, eighth, and ninth spaces in the left axilla. The pain is increased by taking a deep breath, and becomes exceedingly bad in a paroxysm of coughing.

No superficial tenderness, but marked tenderness on pressure or percussion over a patch the size of the palm of the hand in the seventh, eighth, and ninth spaces (fig. 14). No headache. No scalp tenderness.

Both sides of the chest move equally. Percussion note somewhat hyper-resonant. Breath sounds everywhere weak, but expiration is not prolonged. Vocal resonance everywhere feeble. A few scattered râles are heard over the lower part of the left lung.

In the left axilla marked rough friction is heard, extending over the seventh, eighth, and ninth spaces. The area within which friction is heard almost exactly corresponds to the area of deep tenderness (fig. 14).

No other signs or symptoms of importance. Temperature not raised.

December 26.—Friction is less marked. All pain has practically gone, except when he takes a very deep breath. The area of deep tenderness is contracting, and is less marked.

January 3.—All pain and deep tenderness gone.

Sometimes an infarct in the lung will cause a small patch of pleurisy if situated near the surface. Such cases form exquisite examples of the localising value of deep percussion tenderness. I have several times had the opportunity of verifying the position of the infarct which caused this local tenderness. In Case 61 both infarcts were diagnosed during life, and verified after death.

Case 61.—To illustrate the deep tenderness sometimes present when infarcts of the lung cause small patches of pleurisy. Emma D. (V.P.H., Dr. Harris); aged 47; nurse.

At the age of 14 years she had chorea, "due to a fright." It lasted fifteen months. At no time in her life has she had anything resembling acute rheumatism.

She had been working very hard as a sick nurse, and on December 10, 1893, began to feel very tired, hot, and feverish. December 11 her temperature was 104°. She was in bed for a fortnight, with a varying temperature, and then got up. January 10, 1896, the temperature rose again, and she felt very ill.

On admission (January 18, 1896).—She is a small, used-up looking woman of 47. Arcus senilis beginning to appear in both eyes. Face of a grey, dusky pallor.

Respiration rapid. Very short of breath. Some orthopnoea.

Directly after admission she began to complain of pain in the *right* axilla in the sixth, seventh, and eighth spaces. There was no superficial tenderness, but exceedingly marked deep tenderness in the sixth, seventh, and eighth spaces in the mid-axilla.

January 19.—The percussion note is diminished over the lower axilla on the right side, extending back to the line of the angle of the scapula. Over this area the breathing is tubular, and the vocal resonance increased. Over the area of deep tenderness in sixth, seventh, and eighth spaces, coarse friction is audible.

Elsewhere lungs are normal.

Pulse 88, and irregular in rate and force. The pulse is small, but hard.

Apex beat in fifth space and nipple line. Epigastric pulsation. No thrill. The first sound at the apex is sharp, and the second re-duplicated. Pulmonary second sound accentuated. No murmur heard at first.

Liver and spleen not felt.

Urine showed a faint cloud of albumen.

January 20.—She began to spit up red or purple-coloured sputum.

January 22.—All deep tenderness has gone; but friction was occasionally heard over the same area.

January 23.—She began to complain of pain in the *left* axilla, situated in the seventh and eighth spaces in the mid-axilla.

There is exceedingly well marked deep tenderness in the seventh and eighth spaces in the mid-axilla, but no superficial tenderness anywhere.

On the night of January 23, friction appeared over the area in the left axilla.

The diagnosis was made of an infarct in the right lung, followed by a second infarct in the left lung; both reaching the surface, and causing slight pleurisy.

February 20.—She suddenly became hemiplegic, without loss of consciousness.

She then gradually got weaker, and died on March 20.

Post-mortem.—Before the examination was begun, the areas of deep tenderness in the right and left axilla were marked out upon the skin. Dr. Fyffe made a careful dissection, and found two infarcts, as follows. In the right lower lobe was an infarct, a little more than an inch across the base, which had caused thickening of the pleura in its immediate neighbourhood, and this area of thickening lay under the area marked out on the surface. A similar recent infarct, causing a patch of pleurisy, was found in the base of the left lung, and this infarct also lay under the area marked out in the left axilla.

Mitral orifice much stenosed, admitting only one finger. About the edge of the valves were some recent vegetations, and a small erosion. Other valves normal. Hypertrophy and dilatation of left ventricle. No micro-organisms could be found in the valves.

Liver and spleen enlarged.

In the brain an infarct was found, causing softening of part of the internal capsule.

(B) *Referred Pain in Pulmonary Disease.*

The second type of pain met with in diseases of the lungs is that which is usually known as referred pain. It is of no direct localising value, but is situated at certain spots on the chest, or may appear to run round the body at various levels. In these spots we recognise the maximum points of certain areas connected with the central arrangements of the nervous system. Pains of this type are usually described as "intercostal neuralgia," "myalgia," "intercostal rheumatism"—names which are either meaningless, or else imply a false pathology. For these pains do not follow the course of the intercostal nerves, but occupy part of the territory of one nerve, and part of that of another. They are said to be "stabbing," "catching," "aching," or "tight;" and in some cases the patient complains of a true girdle sensation, with a feeling as if his chest were tied in at a certain level. The pain is not usually increased, to any marked extent, by each act of inspiration, but is markedly increased by anything that throws increased work upon the diseased lung. Thus the dyspnoea produced by going up a flight of stairs, will frequently bring on, or increase, referred pain, although each respiratory act is not peculiarly associated with an increase of pain.

Such referred pains are associated with tenderness of the superficial structures of the chest. Now it has been long known that phthisical patients are subject to soreness and tenderness of the chest walls, but this tenderness has been considered of little importance. Walshe¹ noticed this superficial tenderness, and points out that "firm pressure over a broad surface gives relief." Valleix² pointed out that what he called intercostal neuralgia, was accompanied by three tender points: one behind, one in the axilla, and one on the front of the chest. This observation, Walshe confirms; and

¹Third Edition, p. 192.

²"*Traité des Névralgies.*"

points out that no one intercostal nerve is affected, "the pain usually involves the nerves from the sixth to the ninth—in rare instances a single trunk only."

But, in spite of the common neglect of superficial tenderness by the physician, this feature of the disease is well recognised by many patients. They complain that the neck, chest, back, or upper abdomen feels sore and tender, as if it had been beaten or was bruised. They tell you that they have been obliged to loosen their clothes, to wear soft things next the skin; or, in the case of women, that they have been obliged to loosen or remove their corsets. On picking up the superficial structures between the finger and thumb, areas are found, within which the patient complains that the manipulation, however gentle, causes pain. By using the blunt head of a pin these areas can be defined; for the patient either complains that certain parts are sore when touched with the head of the pin, or else he experiences a sensation as if he were being pricked. I must, however, here warn the observer of the fallacies which are attached to the use of the head of the pin upon the anterior and lateral aspects of the chest. He must be very careful, in these regions, that the head of the pin does not impinge on the edge of a rib; for this will cause pain even in a normal individual. Thus I always prefer to lift the skin between the finger and thumb when testing areas on the chest, excepting over such parts, at the breasts,¹ where the pin is obligatory. Here, however, the ribs are so well covered that fallacious results are less likely to be obtained with the head of a pin.

Another fact which shows how purely such pain and tenderness depend upon impulses passing into the central nervous system, is the association of such referred pain on the chest, with areas of referred pain and tenderness on the scalp. This association takes place according to definite rules, which I have laid down in Part II.² of this investigation;

¹ In *multiparæ* the superficial structures of the breast are comparatively insensitive; and superficial tenderness that is well marked over the back and rest of the chest may frequently be almost absent over the breasts, although the breast lies within the tender areas.

² BRAIN, 1894, p. 436.

and the occurrence of such scalp tenderness in diseases of the lungs will be repeatedly alluded to in subsequent sections of this chapter.

§ 2. *Conditions necessary to the production of referred pain in diseases of the lungs.*

Referred pain is produced by distention of a sensitive organ from within, or by the exertion upon it of some tearing or rending strain from without. Thus, if the intestine is distended, referred pain may result. If, on the other hand, it is cut with a sharp knife, as in the second stage of inguinal colotomy, no pain is produced; but if, in the process, it is torn or pulled upon, the patient complains of pain. Moreover, it is essential that, at any rate, some of the end organs within the diseased area should be intact, in order that referred pain may result. Thus no true referred pain and superficial tenderness result from a tooth in which the pulp is dead, although much local pain may be caused by inflammation around the root. Thus any disease which prevents all movement in the diseased organ, or any disease that rapidly destroys its tissues, including the nerve endings, will be unaccompanied by referred pain.

Now, in acute lobar pneumonia, the essential process of the disease is a consolidation of one or more lobes of the lung, with complete obliteration of the alveoli by exudation. The air no longer enters the alveoli of the whole consolidated lobe. But, in spite of its profoundly diseased condition, such a consolidated lobe gives rise to no referred pain, for the whole of the end organs are removed from the influence of pressure from within, or traction from without, by this consolidation. Thus the large majority of cases of acute lobar pneumonia exhibit no referred pain or superficial tenderness, and associated with this absence of referred pain there is no referred headache, or tenderness of the scalp. But in a considerable number of cases of acute lobar pneumonia, the consolidation of the lung is associated with pleurisy of greater or less extent. To this is due the local pain, frequently accompanied by tenderness on percussion or pressure, which is not

an uncommon feature of this disease. I give an instance in which both lower lobes were consolidated; but the only pain that was present was localised over the left base, where marked friction was audible.

Case 62.—To illustrate the absence of referred pain and superficial tenderness in Acute Lobar Pneumonia, and the dependence of the pain frequently present in this disease on coincident pleurisy. Luke L. (V.P.H., Dr. Thorowgood); aged 38; Wood carver.

On the evening of March 29, 1894, he was seized with cold shivers. The next day pain came on in the left side.

On admission (April 3).—Well-built man. Face of a pinkish-yellow colour, with a patch of herpes about the right, lower lip, extending outwards to the cheek. No marked cyanosis.

Respiration 24. Sterno-mastoids acting slightly. Some subjective dyspnoea. He can lie on both sides, but prefers to lie on the left side. Short paroxysms of cough. At infrequent intervals he spits up a small quantity of greenish-brown, frothy expectoration.

He complains of pain over the middle of the left axilla in the seventh, eighth, and ninth interspaces. No superficial tenderness, but extremely well-marked tenderness on percussion, or deep pressure was evoked in the sixth, seventh, eighth, and ninth spaces from about an inch from the costal margin below to the line of the latissimus dorsi above.

No headache. No scalp tenderness.

The percussion note in the left axilla, and below the level of the sixth spine on the left side, is absolutely dull and resistant. Over this area the breath sounds and vocal resonance are almost completely abolished, and in the sixth, seventh, eighth and ninth spaces in the axilla, marked friction is audible.

The percussion note over the right base and in the right axilla is tubular. Over this area the heart sounds are weak, and the vocal resonance fair. Numerous fine crepitations are heard, but no friction is audible.

The temperature was 102.8° F. (39.3° C.) The pulse was good. The urine contained no albumen.

All pain and deep tenderness rapidly disappeared, and on May 6 the temperature was absolutely normal; and the lungs presented no abnormal signs, except a very slight diminution of note, with weak breathing and vocal resonance below the angle of the left scapula.

Thus the form assumed by the disease is all important in determining the presence or absence of pain in the course of phthisis and other diseases of the lungs.

Three stages—*invasion, consolidation, and excavation*

—are usually described for tubercular phthisis; but experience shows that they occupy a very variable amount of the field in the clinical picture of any one case. Thus, in the one extreme type, the patient scarcely complains of anything beyond a cough in the mornings, and a feeling that he is not up to the mark. And yet, when he seeks advice, marked signs of consolidation, and, perhaps, of excavation, are present over the upper part of one lung. The temperature may be very irregular—greatly raised one night and scarcely raised at all the next. The patient may gain weight under treatment, and take his food well; but in spite of this, the cavitation may steadily advance. For whilst, at first, the signs of a cavity may extend to the level of the third dorsal spine, in two or three months similar signs may be present as low as the sixth spine. Yet during this time, all referred pain, referred headache, tenderness of the scalp, and superficial structures of the chest may be entirely absent.

From this point onwards the course followed by the case may be very variable. A sharp rub may make its appearance about the angle of the scapula, or in the axilla; and the whole base of the lung may become adherent; or the upper lobe, or the apex of the lower lobe of the opposite lung, may become rapidly excavated. But neither of these processes is associated with referred pain or superficial tenderness. Thus, in an extreme case of this type, the patient may advance far with the disease without suffering from any referred pain.

Moreover, when excavation, or pleurisy, or an association of both processes, form the main feature in the course of a case of phthisis, the parts which are not visibly affected by tubercle tend to become markedly fibrotic. Thus, in such a case, the adherent, but otherwise unaffected, bases are found, *post-mortem*, to contain excess of connective tissue. They are thus in an unfavourable condition to give rise to referred pain when actually invaded by tubercle.

Thus, what may be called the excavation and pleuritic type of phthisis may run on to the end without showing any referred pain or superficial tenderness.

Case 63.—To illustrate the absence of referred pain and superficial tenderness in those cases of phthisis where excavation is a marked and early feature of the disease.—Herbert K. (V.P.H., Dr. Heron); aged 26; groom.

He was in splendid health until October, 1893. He was then seized with some acute illness. On the second and third days he lay in bed. On the fourth day he got up, and on the fifth day a cough came on, and has not left him since.

He felt languid and ill, but was able to continue work till November. At the end of November he began to sweat at night, and about the beginning of December he began to spit up with his cough. He has lost $7\frac{1}{2}$ lbs. in four months. He has been free from pain throughout.

On admission (January 22, 1894).—Well-built man of 26. Not anæmic. No marked cyanosis. Sweats at night.

Respiration 28. Short of breath on considerable exertion, but not when walking quietly. Cough, single; not very troublesome. Small quantity of expectoration, containing tubercle bacilli.

No pain. No headache. No superficial tenderness anywhere.

The right apex moves less than the left, both in front and behind. The percussion note is diminished on the right side, over the first two spaces in front, and as low as the level of the fourth spine behind. Over this area, both in front and behind, marked cavernous breathing and whispered pectoriloquy are heard, with many metallic râles. A few scattered râles are heard behind over the upper part of the right lung; but the breath sounds at the bases and at the left apex are normal. Larynx, normal. No abnormal signs in heart. Tongue, moist. Appetite, poor. No nausea or vomiting. Bowels, normal. Urine contains no albumen, and is acid.

The temperature rose to 102° F. (39° C.) every night, but fell to normal in the morning.

The signs of cavitation at the right upper part of the right lung spread downwards rapidly, and by March 3 cavernous breathing and whispered pectoriloquy could be heard as low as the level of the sixth spine.

Then sharp, dry friction appeared at the angle of the right scapula, and by April 12 the movement of the right base was much impaired, the note was diminished, and the breath sounds very weak all over this base.

The temperature still remained very high, and he had lost $6\frac{1}{2}$ lbs.

No referred pain, headache, or superficial tenderness appeared throughout the whole time he was under observation.

This case is an extreme instance of phthisis, running a course unattended by referred pain. But the disease may advance in a manner that is so different to the instance I

have just given as to form an almost exactly opposite type. When the patient first seeks advice, there is, perhaps, a little deficiency of movement at one apex. The percussion note is not definitely altered in front or behind, but a few sharp crepitant râles are heard over the upper part of the lung on the side where the movement is deficient. There is a good deal of cough, and some expectoration, in which tubercle bacilli can be found, generally in considerable numbers. The patient may complain of some aching pain about the clavicle, but there may be little or no superficial tenderness. Then the signs rapidly increase, and fine crackling râles may become audible over the upper part of the lung as low as the level of the fourth or fifth dorsal spine, and a few râles and rhonchi may be heard creeping round the vertebral border of the scapula. The breath sounds over the upper part of the affected lung are feeble, and the vocal resonance perhaps a little increased. The patient now complains of referred pain in the affected side of the chest, and more or less superficial tenderness is present both on the chest and on the scalp.

Let us suppose that the left side is first affected. This side may now remain quiet for a time, and, although the lower part of the interscapular fossa may be free from râles, they persist over the upper lobe and apex of the lower lobe of the left lung. All referred pain and superficial tenderness may die away. Then, after a varying interval, a sudden outburst of râles and rhonchi may appear over the right side, extending to the level of the angle of the scapula. Referred pain will now reappear mainly over the right side of the chest and back, accompanied by widespread superficial tenderness, more marked on the right side than the left, but present on both sides of the chest.

The acute signs on the right side may now die away, leaving the upper lobe and apex of the lower lobe damaged, as shown by the weak breath sounds and a few irregular râles. The referred pain and superficial tenderness may either disappear, or be very much diminished in intensity and extent.

The next acute outburst may occur on the same or on

the opposite side, and be accompanied by a further development of referred pain and superficial tenderness of the chest and scalp.

Each such acute outburst subsides, but leaves the lung more damaged than before, and, sooner or later, signs of excavation make their appearance, generally at the upper part of the lung first affected. But for some time after the first signs of excavation have appeared, the progress of the case is characterised by periodic outbursts of râles and rhonchi over the lower portions of the lungs.

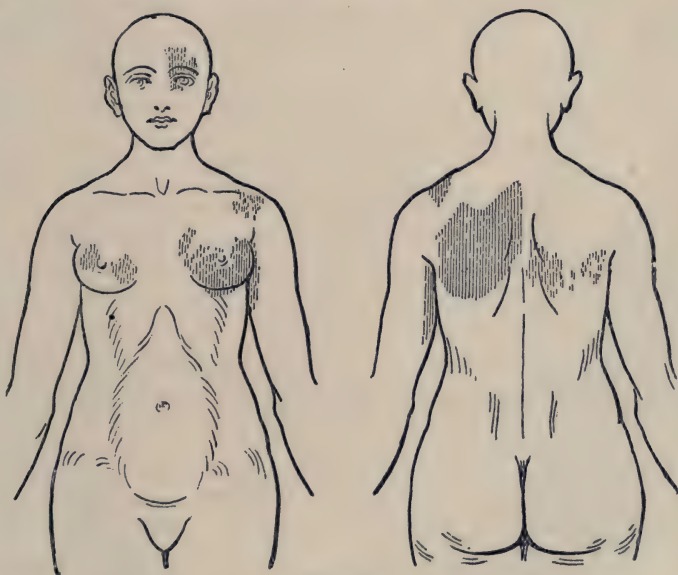


FIG. 15.

To show the superficial tenderness present in a case of phthisis [No. 64] during the implication of the left lung [Jan. 25].

It is this type of phthisis (of which I have given an extreme example in Case No. 64) that is accompanied by referred pain and superficial tenderness in its more exquisite form. The reason for this, I conceive, to be that, during the bronchitic type of invasion, the alveoli are rather encroached upon than primarily affected. The end organs are irritated but not destroyed, and are capable of conveying impressions even from the diseased portions of the lungs.

Case 64.—To illustrate the type of *Phthisis* in which the advance takes place by repeated so-called bronchitic attacks. Catherine C. (V.P.H., Dr. Harris), aged 24; housewife.

About August 1893, she began to have a cough and to spit phlegm, occasionally streaked with blood. She then began to lose flesh. November, 1893, she began to have pain in her left side.

On Admission (December, 15, 1893).—She is a largely-built woman, distinctly wasted. Face pale. No cyanosis. Sweats profusely at night. The temperature was 102° F. (38·9° C.) on the first night. This fell to 98° F. (36·7° C.), in the morning; rising to 100·4° F. (38° C.) the second evening.

Respiration 28. The *alæ nasi* dilate. Some subjective dyspnoea. Cough not very troublesome. Expectoration, in considerable quantity, consists of small masses of muco-purulent material, streaked with blood, and contains tubercle bacilli.

No pain. No headache. No superficial tenderness.

The movement at the apices is somewhat poor, and the right base moves rather more freely than the left. There is no very marked difference in the percussion note over the apices, but the note over the left apex in front is perhaps a little less resonant than over the right. On the left side, in the first and second interspaces in front, and as low as the second spine behind, the breath sounds are weak, vocal resonance increased, and numerous crackling râles are audible. Otherwise there are no markedly abnormal signs in the lungs.

Pulse 94. Small stroke and regular. Heart sounds normal. Tongue moist, and a little grey; papillæ prominent, not actually furred. Appetite good. Bowels irregular. No nausea. No vomiting. No pain after food. Liver and spleen not felt. Urine acid; no albumen, nor sugar.

January 14.—The temperature ranged to 101° F. (38·3° C.) every night during the first month. She has had very slight referred pains, but now no definite superficial tenderness could be found.

The signs at the left apex have increased. There is no distinct diminution of note over the upper two spaces on the left side in front. Over this area the breath sounds are feeble, expiration distinctly prolonged, and vocal resonance is markedly increased. Numerous fine crackling râles are heard as low as the third rib.

Behind, on the left side, the note is diminished as low as the level of the fourth spine. The breath sounds are exceedingly feeble, and the vocal resonance is a little increased. Fine crackling râles are heard to the level of the fourth spine behind.

No abnormal signs on the right side.

January 25.—She complains of shooting pains in the left side of the chest and left half of forehead, accompanied by superficial tenderness, as in fig. 15.

The cough is not more troublesome, nor the expectoration more profuse. The crackling râles at the upper part of the left

lung are more numerous, and extend down to the level of the sixth spine behind. When the left hand is placed on the right shoulder, these crackling râles are heard along the vertebral border of the scapula, but when the arm is dropped to the side they are not heard at the angle of the scapula.

January 30.—The signs on the left side of the chest have extended a little lower, but have not materially altered. A sudden outburst has, however, taken place on the *right* side, and crackling

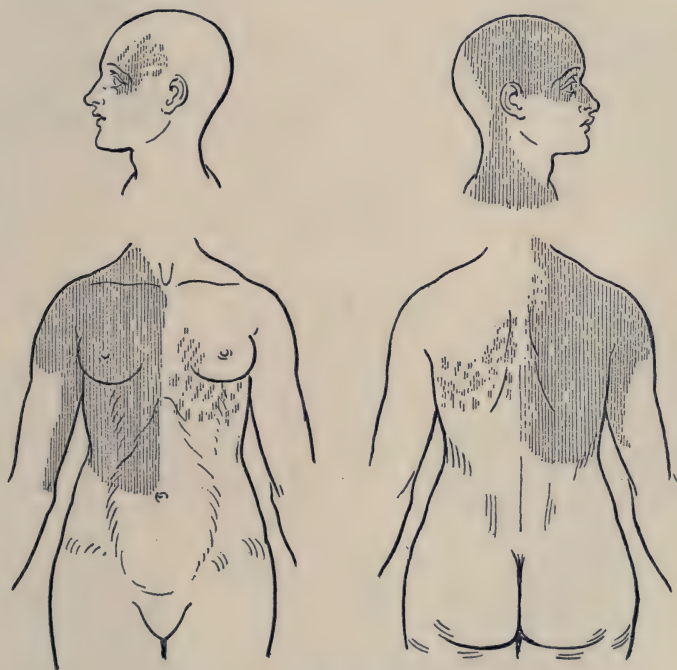


FIG. 16.

To show the superficial tenderness present in the same case of phthisis [No. 64], in a later stage, during the implication of the right lung [Jan. 30].

râles are heard over the whole upper part of the inter-scapular fossa, and about the angle of the right scapula.

The pain has shifted. She complains now of much pain all down the right side, and over the right half of the head. Extremely widespread tenderness is present over the right side of the body and scalp, such as not infrequently accompanies an acute outburst of this kind. (Fig. 16).

February 15.—She is now complaining of pain over the epigastrium, lower ribs, and under the shoulder blades behind. The

headache is mainly on the top of her head. Marked superficial tenderness as in fig. 17.

The appetite is poor. The tongue is clean. Bowels opened daily. No diarrhoea or constipation now. She is beginning to feel a little sick, but has not vomited.

On the left side in front, the dulness extends to the fifth rib. Cavernous breathing and whispered pectoriloquy are heard in the upper two spaces. Behind the second space the heart sounds are very weak, and the vocal resonance increased. Numerous râles audible.

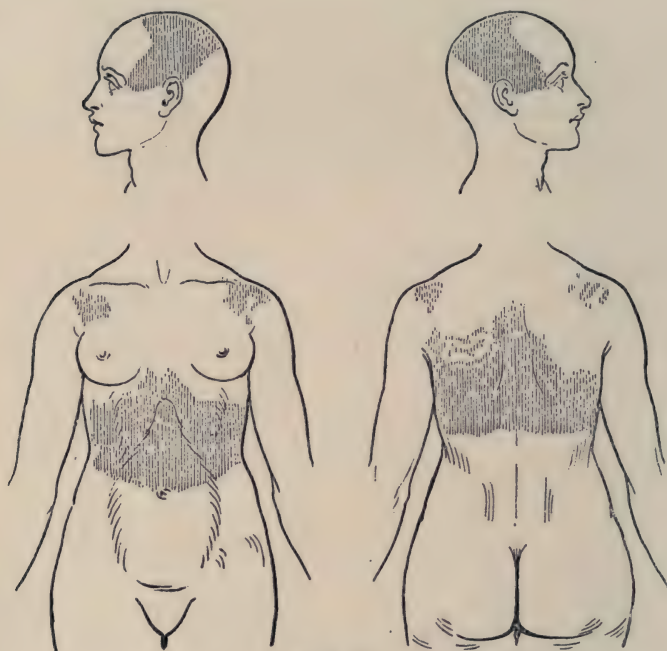


FIG. 17.

To show the superficial tenderness present during a later stage of the same case [No. 64].

Over the upper part of the left lung behind, the note is dull as low as the sixth space. The breath sounds over the apex are very weak, but expiration is hollow, and bronchophony is heard. Behind this to the base the breath sounds are very feeble, expiration is prolonged, and vocal resonance is increased. Râles are heard behind from the apex to the level of the eighth spine.

On the right side in front, râles are heard to the level of the fifth spine. Behind the note is somewhat diminished, but less so than on the left side. The breath sounds are feeble, and

expiration is prolonged as low as the angle of the scapula, and over the whole of this area, vocal resonance is increased, and numerous râles are heard.

Thus both bases are now being steadily affected.

She remained in this stage till March 11, when another outburst took place over the whole right lower lobe, especially about the angle of the right scapula, and in the right axilla.

This produced another widespread outburst of superficial tenderness of both sides of the body and scalp.

The temperature now rose to 103° F. (39.5° C.) every night, and she became very weak.

Thus, in the space of three months, the march of the disease was as follows :—Firstly, affection of the left apex, not associated with referred pain. Secondly, spread along the upper border of the left lower lobe (fig. 15). Thirdly, affection of the apex of the right lower lobe, together with an acute outburst within this lobe (fig. 16). Fourthly, steady advance down both sides within the lower lobe (fig. 17). Fifthly, an acute outburst over the whole of the right lower lobe, from which it never even partially recovered.

Between these two extreme types of invasion and progress innumerable variations occur, but the more nearly the course of a case of phthisis approaches the bronchitic type, the more certainly will referred pain and superficial tenderness tend to appear, and form a feature in the clinical picture. The more nearly it approaches the excavation and pleuritic type, the less likely are referred pain and superficial tenderness to make their appearance.

Thus, when considering the origin of referred pain in any particular case of phthisis, many different factors have to be taken into account. No referred pain accompanies rapid and absolute consolidation of one lobe, or its subsequent excavation. On the other hand, partial implication of the lung by multiple foci of disease [not acute miliary tubercles] scattered amongst relatively healthy tissue, or the gradual opening out of a collapsed or airless patch, are particularly liable to be associated with referred pain. Thus, the physical signs which most frequently stand in connection with the development of referred pain in the course of a case of phthisis, are small sharp, dry râles and sibilant rhonchi. But these small râles may be present for months over some particular area of the chest without there being any referred pain. Thus, it is not the quality nor the nature of the

adventitious sounds which is important, but the sudden appearance of these small râles and sibilant rhonchi over parts where they have not been audible before. The larger and moister râles are not usually associated with referred pain.

§ 3. *An attempt to connect the presence of certain areas of superficial tenderness with disease of certain portions of the lung.*

Now that we have considered the conditions and physical manifestations, which are associated with referred pain in the course of a case of phthisis, it remains to be seen how far such referred pain represents the position of the disease.

At the outset, we are met by the difficulty that every referred pain, when sufficiently intense, or of sufficiently long duration, passes over and affects the same areas on the opposite side of the body. Thus, if the patient complains of pain under the shoulder blades and across the lower part of the chest, we have to enquire on which side the pain began, and on which side it is worse. Both, however, depend upon the patient's memory and observation, and the answers are, therefore, of but little use in practice. Far more valuable is an examination of the condition of the superficial tenderness on the two sides of the body. If the tenderness on the one side is very marked, and extends completely round the body from the middle line behind to the middle line in front; whilst on the opposite side the intensity is less, and the distribution spotty, and only the maximum points well marked, the referred pain probably began over the former and spread to the latter side. Sometimes the headache and scalp tenderness afford a guide to the side upon which the disturbance began, for the headache and scalp tenderness are usually more marked upon that side upon which the pain on the body is most marked.

By intensity of tenderness, I do not mean an expression of the patient's opinion as to which side is the most tender. I judge of the intensity of the tenderness (1) by the sensation produced by the blunt head of a pin. Stimulation with the

blunt head of a pin may only cause a feeling of soreness over the tender area, if the tenderness is but slight, or, if the tenderness is more acute, the patient may complain that he is being pricked or stabbed. Thus, in the minor degree of tenderness, the patient recognises a change in the sensibility of his skin, whilst in the major degree he complains of a change in the nature of the stimulus. (2) By the completeness with which the bands of tenderness are represented on the surface of the chest. (3) By the exaggeration of the superficial reflexes.

But another disturbing factor in the attempt to determine the localising value of referred pain in phthisis lies in the fact that a fresh outbreak in one part of the lung is usually associated with a rise of temperature. Now, wherever referred pain and tenderness are present, and a sudden rise of temperature occurs, the pain, and particularly the tenderness, tend to spread widely. Thus a sudden acute outburst over the upper part of one lower lobe of the lung will not unfrequently lead to exceedingly widespread tenderness of the neck, trunk, and scalp, mainly on the side of the fresh lesion, but also on the opposite side. But though a rise of temperature tends to cause spreading in those cases where referred pain and tenderness are already present, it does not tend to generate tenderness in those cases where it would not otherwise appear. Thus where the lesion consists of a large cavity, the temperature is frequently raised, and even a rigor may occur; but no referred pain or superficial tenderness of the body or scalp appear.

A third difficulty that meets us is the tendency to spreading, and the consequent diminution in the localising value of the referred pain, produced by the cachexia which accompanies the disease. Any referred pain and tenderness which occurs in phthisis tends to spread, owing to the deficient resistance within the nervous system, produced by the phthisical cachexia. Thus the referred pain and superficial tenderness produced by the inflammation of the pulp of a tooth will tend to spread beyond the limits usual when that particular tooth is affected. Hypermetropia headaches

also tend to become much more troublesome, and the tenderness to spread beyond the forehead, in consequence of the patient's cachectic condition,

Case 65.—To illustrate the tendency to spread shown by all referred pain, of whatever origin, owing to the cachexia of phthisis. Sarah Ann T. (V.P.H., Dr. Sainsbury).

Within the last six months she has had cold after cold, and repeatedly lost her voice. She has grown much thinner, and sweated at nights throughout this period. Slight hæmoptysis a month before admission.

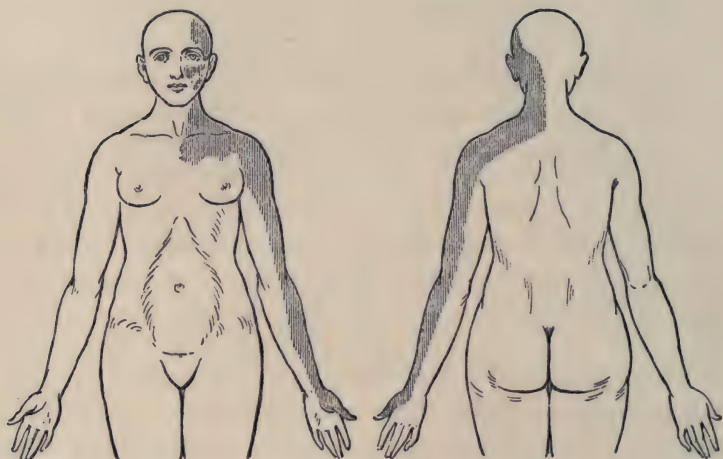


FIG. 18.

To show the wide-spread tenderness produced by disease of the pulp of the second lower molar [Case No. 65]. The great extent of this tenderness is probably due to the cachexia produced by phthisis.

I will not enter into great detail, for this case is quoted to show how any visceral pain tends to spread in phthisis whatever may be its origin.

Dulness was present over the right side behind to the level of the sixth spine. Bronchial breathing and pectoriloquy were heard over the upper part of this area, and a considerable number of râles were audible. There was a small ulcer on the left vocal cord.

She remained free from pain till five weeks after admission, when she developed pain in the second lower molar on the left side. The pulp cavity was exposed. Neuralgia came on at the same time in the face, with pain in the left ear. Then all the

teeth in the left half of the lower jaw up to the canine began to ache. The whole left side of the face, neck, and a part of the left arm began to ache. She feels as if all the power of lifting her left arm were gone, but she can use her fingers perfectly.

There is extensively widespread superficial tenderness over the left side of her face, neck, and radial side of left arm. (Fig. 18).

The temperature was 100·2° F. (37·9° C.).

The whole disappeared within four days of removing the second lower molar tooth.

Now such a widespread disturbance is not usual,¹ except where the patient is anæmic and cachectic. This case shows how the cachexia of phthisis leads to a condition in which referred pain spreads with great rapidity.

Bearing in mind these sources of fallacy, the referred pain and superficial tenderness appear on the same side of the body and scalp as the lesion in the lung.

An attempt to further differentiate the referred pain and superficial tenderness which occur in phthisis is met by a difficulty in nomenclature. To say that certain referred pain is due to an affection of the upper lobe, and pain in another position is associated with affection of the lower lobes would be simple enough. But the upper lobe of one lung is rarely affected apart from the apex of the lower lobe, and thus a statement in this form would be both unpractical and indefinite. Again, from the point of view of the advance of the disease, the lower lobe is made up of several portions. For, as Fowler² showed in his admirable paper, the apex of the lower lobe is easily affected in the course of tuberculous phthisis, and the disease then progresses along the upper and outer portion of the lower lobe beneath the interlobular septum. Spreading later to the other portions of the lower lobe of the lung.

Again, the affection of the lower lobe is more often the cause of referred pain than the upper lobe. For the ordinary process of destruction in the upper lobe is one of consolidation followed by excavation. But the mode of extension towards the base is not usually by an advancing line of

¹ *Vide* part ii. of this investigation, BRAIN, 1894, p. 414, and p. 361, fig. 10.

² *The Localisation of the Lesions of Phthisis*. Kingston Fowler. London, 1888.

consolidation, but by scattered nodules of infiltration often arranged in a racemose manner. Now this is just the condition which is likely to lead to referred pain and superficial tenderness, and thus the implication of the lower lobes is far more commonly the cause of referred pain in phthisis than that of the upper lobes.

Another difficulty that meets us at the outset is familiar to everyone who has performed many *post-mortem* examinations. The amount of the lesion discovered after death is frequently considerably in excess of the signs during life. Thus all the following attempts to show that certain portions of the lung are associated with certain segments of the central nervous system must be accepted with a liberal margin for error. Such an attempt at differentiation would be quite impossible if chronic phthisis advanced steadily down the lung. It is only rendered practicable by the fact that it is usually the small acute outbursts in the course of the chronic disease that cause referred pain and superficial tenderness, and that the signs audible during such an outburst, more nearly represent the extent of the disease than the chronic signs present between any two such outbursts of fresh activity.

Purely for the purpose of attempting to associate the extent of the pulmonary lesion in phthisis, with the areas of superficial tenderness on the chest, I have been led to divide the lung into the following parts. I do not suppose these divisions have any permanent value, they are purely rough empirical divisions, by which the commoner lines of spread may be classified for association with areas of referred pain and tenderness in the one disease, *i.e.*, phthisis. These divisions are marked out as follows:—

(1) Place the hand upon the opposite shoulder,¹ so that the angle of the scapula becomes tilted upwards and outwards. Draw a line from the fifth dorsal spine (sixth dorsal vertebra) outwards and slightly downwards to the angle of the tilted scapula. Above this line lies the upper part of the lower lobe.

(2) Let the hand fall to the side. Then draw a line from

¹ Cf. Fowler, *op. cit.*

the seventh dorsal spine outwards and slightly downwards following the line of the eighth rib, and passing below the angle of the scapula. Above this rough line lies the middle division of the lower lobe.

(3) From this line to the base of the lung lies the third division of the lower lobe.

The upper lobe of the lung is but rarely affected without some implication of the apex of the lower lobe. But in those few cases where the disease was very early, and the râles were mainly present in front, and in the supra-clavicular fossa, the pain and superficial tenderness lay mainly within the third and fourth cervical and third dorsal areas. Now, early phthisis is usually said to cause "pain over the lung," or "tenderness over the upper intercostal spaces." But where this pain and tenderness are present, a careful examination shows that the pain is frequently referred and not local, and that the tenderness is superficial. Thus, the tenderness frequently runs up the back of the neck, and is present over parts of the neck under which no lung tissue is situated. In the same way, the little tag of tenderness on the arm belonging to the third dorsal area can frequently be demonstrated at a distance from the lung.

As soon as acute signs become audible in the neighbourhood of the fourth and fifth dorsal spines, and the râles are found creeping round the vertebral border of the scapula when the hand is placed on the opposite shoulder, the third, fourth, and fifth dorsal areas tend to make their appearance.

When the disease attacks the middle division of the lower lobe, and the râles are found clustering about the angle of the scapula (with the arm by the side) and extend into the lower part of the axilla, the fifth, sixth and seventh, but particularly the sixth dorsal areas, make their appearance.

When the lowest division of the base of the lung becomes affected, the tenderness lies mainly over the seventh, eighth, and ninth dorsal areas, but most commonly over the seventh and eighth in phthisis.

I have entirely neglected the middle lobe of the right lung, because it is almost impossible to localise the disease

affecting it, even with the roughness that is alone possible elsewhere. Moreover, it is frequently found, *post mortem*, to have escaped, although the greater part of the right lung is affected.

The lung is, therefore, associated with the third and fourth cervical, and the third to the ninth dorsal areas inclusive. The second dorsal escapes, as a rule, except in such cases where we have reason to suspect that the tenderness has spread. Sometimes, however, it seems to make its appearance even where such spread seems improbable. The possibility of variation must always be borne in mind.

In my first paper, I did not place the supply of the lung lower than the fifth dorsal segment; for from this point downwards we reach the gastric areas, and at that stage of the research, I did not feel justified in associating any pain and tenderness accompanied by gastric disturbances with lesion in the lung. My present opinion on this matter is embodied in §4 of this chapter.

As lesions of the upper part of the lung are associated with pain and tenderness within the third and fourth cervical and upper dorsal areas, they are accompanied by superficial tenderness over the forehead and around the eyes. On the other hand, as soon as the lower lobes are considerably affected, the scalp tenderness will travel back to the temporal, vertical, or even to the parietal regions. The lower the lesion in the lung, the nearer the tenderness over the body approaches the ninth dorsal area, and the further back is the tenderness on the scalp.

To summarise the conclusions to which we have arrived in this section :—

(1) All referred pain and superficial tenderness which makes its appearance, from whatever cause, in the course of subacute phthisis tends to spread widely. This is probably due to (A) the cachexia, and (B) the temperature which accompanies the disease.

(2) Referred pain produced by the disease of the lung was most marked, and of greatest intensity on the same side of the trunk as the fresh lesion. Both pain and tenderness may, however, secondarily make their appearance on the

opposite side of the lung, but appear later, disappear earlier, and are not of the same intensity as on the side of the lesion.

(3) The innervation of the lung is connected with the third and fourth cervical segmental areas, and with all the dorsal segmental areas from the third to the ninth. The lower lobe of the lung is particularly connected with the dorsal areas, especially from the fifth dorsal downwards to the ninth.

§ 4. *Gastric Disturbances in the course of Phthisis.*

We have just seen that referred pain and superficial tenderness appear over the middle dorsal areas when the bases of the lungs become affected with disease of a certain type. But these areas, from the sixth to the ninth dorsal, stand in close relation with the stomach, especially those on the left side. Thus two organs, the bases of the lungs and the stomach, send sensory impulses into the same segments of the central nervous system. Now, when any two organs send impulses into the same segments of the central nervous system, an affection of the one which causes referred pain and superficial tenderness, tends to cause a reflex disturbance in the other organ that refers into the same areas. For instance, an inflammation of the pulp of the wisdom tooth of the lower jaw causes a soreness of the fauces and a sense of pain and swelling on the same side as the diseased tooth. Glaucoma will set those teeth aching which refer into the same areas that have become tender owing to the rise of tension in the globe. Thus, it is not surprising that the sudden appearance of referred pain and tenderness over the sixth to the ninth dorsal areas, in consequence of a fresh outburst in the lung, should be accompanied by symptoms referred to the gastro-intestinal tract.

But tubercular phthisis is in itself associated with gastric symptoms, and it thus becomes very difficult to say whether the pain, tenderness, nausea, and vomiting are, in reality due to the fresh affection of the lung, or to some intercurrent gastric attack. In some cases, however, the gastric dis-

turbance seems to be clearly secondary to the fresh outburst in the lung.

The dyspepsia which may occur in the course of tubercular phthisis can be divided clinically into three groups:—

(1) The *prodromal* dyspepsia which occurs during a period of the disease when the signs in the lungs are either absent, or exceedingly slight.

(2) The dyspepsia of *invasion*, which occurs during the gradual invasion of the lung by the tubercular disease.

(3) The dyspepsia of *dissolution*, occurring in the last stages of the disease.

Now, the dyspepsia of dissolution is very rarely accompanied by referred pain and superficial tenderness. It is mainly characterised by flatulence, eructations, and signs of dilatation of the stomach, and feeble motor and sensory activity. Thus, this form of gastric disturbance does not come within the scope of this enquiry, owing to the fact that it does not cause referred pain and superficial tenderness.

The prodromal dyspepsia, on the other hand, is accompanied by marked and widespread referred pain and superficial tenderness. But at the time it occurs the signs in the lungs are either absent, or exceedingly slight; and thus it is certainly not due to what may be called sympathetic reference. It seems to stand in relation with the cachexia which signalises the onset of tubercle of the lungs in the same way that gastralgia so frequently accompanies another general state—anæmia. I do not mean to say that this state is an anæmia, for it differs in certain striking particulars from a true anæmia, and I, therefore, prefer to speak of it as a cachexia. Now there are certain general states, which in themselves seem to be peculiarly associated with, more or less, widespread referred pain and superficial tenderness. To these general states belong the prephthisical cachexia, and certain anæmias, and I shall, therefore, postpone the full consideration of this prodromal dyspepsia, the outcome of the prephthisical cachexia, until I come to treat of these general states. I give the following case (No. 66) as an instance of this form of dyspepsia.

Case 66.—To illustrate the referred pain and superficial tenderness sometimes associated with the prodromal dyspepsia of phthisis.—Janet N. (V.P.H., Dr. Sainsbury).

January, 1887.—At the age of 15, she was admitted to V.P.H., complaining of loss of appetite and weakness. She had not begun to menstruate. She was then pale and thin. She vomited her food as soon as she took it, and had much pain at the epigastrium, increased by taking food. The abdominal walls were

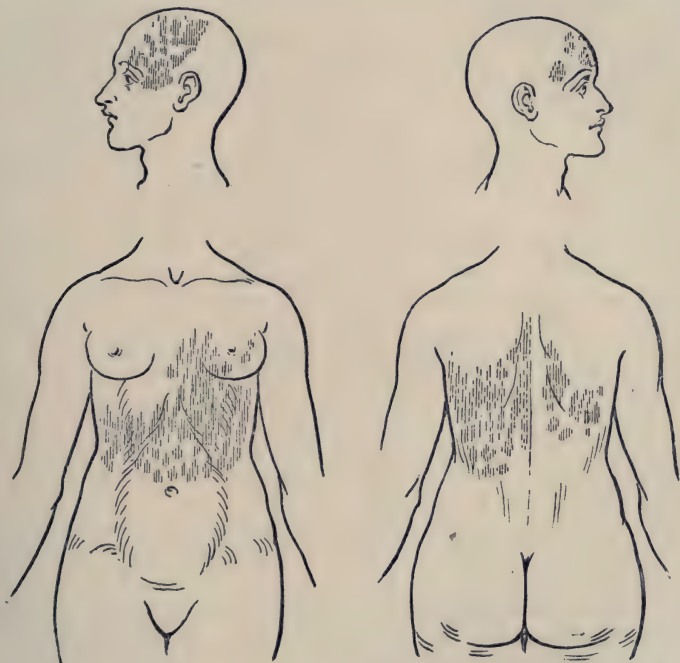


FIG. 19.

To show the superficial tenderness present during the course of the prodromal dyspepsia in Case No. 66.

hyper-æsthetic. Tongue, red and raw looking. Teeth, good. A systolic (hæmic) murmur was heard over the upper part of the sternum; the pulmonary second sound was increased. The temperature was normal throughout.

She left the hospital much improved, but throughout the next four years had several similar attacks.

January, 1891.—At the age of 19 she was again admitted. She had now developed a slight hacking cough, which was only followed by expectoration in the early morning. The sputum contained scarcely any purulent material, and Dr. Hadley was

unable to find any tubercle bacilli. She had been losing flesh for three months, and sweated slightly at night. No definite abnormal signs could be found in the lungs. A systolic hæmic murmur was heard over the second left interspace, and a hum was audible in the veins of the neck. The teeth were still good. Tongue, red. Appetite, bad. Suffered a good deal from flatulence.

February 10.—The tonsils swelled, and the lymphatic glands in the neck became enlarged. The gland in the neck remained enlarged for a considerable time.

She much improved, but in July, 1893, began to feel tired and faint, and to suffer from nausea, whether she took food or not. Vomiting now returned, and she again began to suffer from a slight cough.

October 24, 1893.—She was again admitted to V.P.H. She was now 21, but looked older. The face was pale, of a somewhat opaque white, with a slight flush on each cheek. The ankles swell at the end of the day, but there is no œdema when she is kept in bed. She sweats slightly at night. She was fairly well nourished, and showed no signs of wasting.

She complained of palpitation and shortness of breath. No objective dyspnoea when in bed. Slight single cough, especially in the morning. Expectoration scanty, consisting of a few muco-purulent lumps, in which no tubercle bacilli could be found. She suffers from pain in the epigastrium and back, accompanied by considerable superficial tenderness as in fig. 19. She complains mainly of bitemporal headache, and this headache is accompanied by considerable superficial tenderness of the scalp.

The only abnormal physical signs in the chest were slight diminution of movement over the left apex, with very slight diminution of the percussion note. A few crackling sounds were heard in the upper two spaces on the left side in front. Pulse, 72; low tension. Hæmaglobin, 50 per cent. Red corpuscles, 60 per cent. (3,000,000 in a cub. mm.). No hæmic murmur could now be heard in the chest.

The teeth have decayed very rapidly. The upper central and lateral incisors have gone, and are replaced by a well-made plate. The left second bicuspid, first molar, and right second molar, in upper jaw, exist only as stumps. In addition, many teeth in both jaws show signs of caries.

Tongue, cracked transversely, dry in the centre, and red, but not furred. Appetite, bad. Feels sick at varying times after food. She cannot take fat in any form. Vomiting occasionally. Some flatulence. Bowels opened daily; not constipated.

Liver and spleen cannot be felt.

Menstruation began at 17. Irregular, at intervals of one to two months. She menstruated during her stay in hospital, and suffered from considerable pain and widespread superficial tenderness, which gradually disappeared when the menstruation ceased.

The pain generalised two days before the flow came on. The flow lasted two and a-half days, and the amount was scanty.

The temperature was very irregular. It was invariably raised in the evening, but whereas it sometimes only reached 99° F. (37.2° C.), it occasionally ran up to 100.6° F. (38.1° C.), falling again in the morning to 98° F. (36.7° C.).

During a stay of eight weeks in the hospital she gained 7 lbs. in weight, and much improved.

January, 1894.—The cough returned, and she again became weak and languid, but without vomiting. She had been under continuous treatment with iron, and the hæmaglobin is now 100 per cent., and the red corpuscles 97 per cent. (4,860,000). The movement of the left apex is still somewhat deficient, and a few crackling râles have reappeared both in back and in front on the left side.

August, 1894.—She married. When she became pregnant the vomiting and pain came on again, and she was in bed four months.

August, 1895.—The child was born, and from that time the vomiting and gastric symptoms have much improved, but she began to suffer from extreme weakness. She began to sweat at night again.

February, 1896.—She is obviously thin, and has lost a stone in weight. Sweats profusely at night.

Tongue red and moist. Appetite was very good, but she cannot bear fat of any kind. No pain after food. No headache. No nausea. No vomiting. She now takes large quantities of food, and after meals bitter stuff rises into the mouth, but does not cause her any discomfort. A good deal of flatus at all times.

Coughs, especially in the morning. Expectoration, thick and yellow.

Deficiency of movement at left apex, back and front. Note at left apex not good, but note at right apex, back and front, is considerably diminished. Over upper two spaces, on right side in front, breath sounds are weak, and vocal resonance is increased. Over upper part of right lung behind, breathing is bronchial, vocal resonance is increased, and fine crackling râles can be heard as low as the level of the fifth spine.

Thus the signs of phthisis have now become evident, and the whole character of the clinical picture has altered.

There now remains for consideration, a group of cases of phthisis in which referred pain and tenderness make their appearance over the sixth to the ninth dorsal areas, during the course of the invasion of the lung by the disease. The difficulty in such cases lies in tracking the referred pain and tenderness to its fundamental cause, and, in many

cases, it is practically impossible to say whether the lung or the stomach is the organ that gives birth to the referred pain. However, I think, some help in this difficult task can be gained by dividing this *clinical* group into three *physiological* subdivisions. These three subdivisions of the dyspepsia of invasion I will now consider seriatim.

(A) *Pseudo-Gastric Disturbance.*

The patient's general condition may be good, the tongue clean, appetite fair, bowels regular, and referred pain may be absent, superficial tenderness may be absent or only slightly marked, or may, perhaps, be situated entirely over the upper areas of the chest and the front of the scalp. All tenderness over the sixth to the ninth dorsal areas, on the body, and from the fronto-temporal area backwards on the scalp are absent.

Then a slight recrudescence of activity takes place in the lung disease. Fine dry râles, perhaps accompanied by sibilant rhonchus, appear behind over that part of the lung which lies at the level of the seventh dorsal spine. Referred pain accompanied by more or less marked superficial tenderness, appears in one or more of the areas from the sixth to the ninth dorsal, accompanied by scalp tenderness over the temporal, vertical, or parietal areas. The outburst of fresh activity in the lung will probably be accompanied by some rise of temperature, and thus the pain and superficial tenderness, though more marked on the side of the affected lung, will tend to double at the same level of the central nervous system; this is peculiarly liable to happen on the scalp.

But the disturbance of any organ, whether of the head or trunk, which causes well-marked bilateral referred pain and superficial tenderness of the temporal region is associated with nausea, loss of appetite, and possibly, vomiting. Thus, this nausea, and even vomiting, can be excited by affections of the eye, although there is no tenderness over the gastric areas of the trunk.

Thus, when a slight recrudescence of activity occurs in that part of the lung which lies below the angle of the

scapula, the patient may complain, not only of referred pain and tenderness over the gastric areas of the trunk and temporal region of the scalp, but also of loss of appetite and nausea; later, actual vomiting may occur. The tongue is at first quite unaltered, the bowels remain regular, and the evacuations are unchanged in character, the pain is not increased by food, and vomiting does not necessarily relieve the pain and headache.

On the other hand, anything which relieves the pain and headache, will remove the nausea and vomiting. Thus, mustard leaves applied to the maximum spots of the affected areas of the chest or back, or a dose of phenacetin (which acts more quickly on the headache, but also on the referred pain of the trunk) will remove the nausea and vomiting in this mild and purely reflex type of gastric disturbance.

Such a condition rarely lasts long, for, if it does not pass off within forty-eight hours, it tends to merge into the second form next to be considered.

I might have selected a case of ordinary chronic phthisis as my example of this form of gastric disturbance, I have, however, chosen case No. 67, on account of the shortness of the attack of pain and headache, and the complete return of appetite, with the cessation of the nausea and vomiting.

Case 67.—To illustrate the first, or reflex type, of gastric disturbance which occurs in the course of phthisis. Ada W. (V.P.H., Dr. Harris), aged 17. Admitted, August 17, 1894.

When a child she had a fever, about which no details can be obtained. Since then she has always been under a doctor for her chest. Her cough has become worse during the last five years.

She is a small girl of 17, but looks much older. She is ill-developed, and the fingers are markedly clubbed. There is no wasting, the body is well covered, and the breasts well formed. She does not sweat at night. There is a marked lateral curvature with the concavity towards the left in the mid-dorsal region.

Respiration 24. No objective or subjective dyspnoea, except when she exerts herself. The dyspnoea then becomes marked. Sometimes she does not cough throughout the day. Then, on first waking, she has a paroxysm of coughing, in which she retches, bringing up a large quantity of greenish-yellow masses of stale smelling muco-pus. Many most careful examinations failed to show any tubercle bacilli.

She is usually free from pain and superficial tenderness.

The whole left side is somewhat retracted, and does not move with respiration. Back and front this side is dull. Over the upper part of the left lung the breath sounds are weak, expiration prolonged, and vocal resonance diminished, but below the level of the sixth spine the breath sounds are almost tubular, and bronchophony is audible. These signs extend to the extreme base. From top to bottom on the left side large moist consonating râles are heard. Thus the disease on the left side seems to be old, and the signs did not alter appreciably in three months.

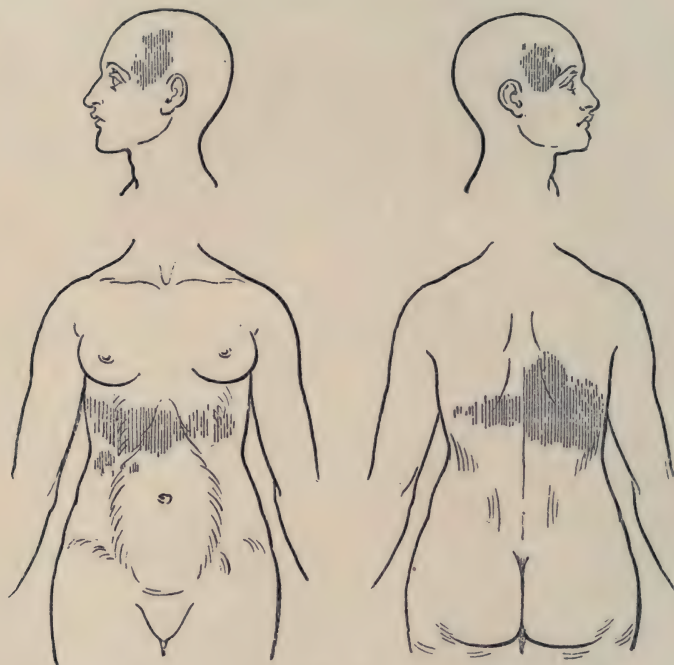


FIG. 20.

To show the areas of tenderness present during an attack of reflex gastric disturbance in Case No. 67.

The right side of the chest is free from abnormal signs, except for a few very large moist râles at the right base.

The pulse was 100, very small, regular, and somewhat hard.

The apex beat seems to be drawn up into the third space on the left side. Heart sounds are normal.

Tongue normal and moist. Appetite good. No pain, nausea, or vomiting (except with the paroxysms of coughing). Bowels normal.

Urine 1015, acid, and containing a considerable quantity of albumen.

She continued in this condition until September 18, when she began to complain of pain under the right shoulder, and in the epigastrium. Some pain on left side, but to a less degree. Bitemporal headache. This pain was associated with marked superficial tenderness, as in fig. 20, both of body and scalp.

She felt very sick, and vomited twice. The appetite was not good, but she could take her food, and the food caused no pain. The pain was always present whether she took food or not. The tongue was clean and moist. The motions were unaltered.

The cough was not increased, and the sputum still had the stale smell, but was not truly offensive.

At the right base the breath sounds are bronchial, the vocal resonance increased, and a large number of râles have made their appearance below the angle of the right scapula.

The temperature which usually only touched 99° F. (37·4 C.), ran up to 101° F. (38·3° C.)

September 19.—By the evening the pain, tenderness, nausea, and vomiting had disappeared. She took her tea with a good appetite. The temperature did not rise above 99·4° F. (37·5° C.)

She had several similar slight attacks, in the course of the three months she was under observation, apparently due to an infection of the comparatively healthy side by the pus from the dilated bronchi of the left lung, which was in a fibroid condition.

(B)—Sympathetic Hyper-æsthesia of the Stomach.

If the referred pain and tenderness over the gastric areas, produced by the condition of the lung, continues for some length of time, or is of marked intensity from the beginning, the stomach seems to become hyper-æsthetic by sympathy. For not only do nausea and occasional vomiting make their appearance but the pain is distinctly increased by the ingestion of food, and is partly relieved by vomiting. Such patients complain that pain is always more or less present, but that it is markedly increased after taking food and, if vomiting occurs, the pain is relieved, and sinks back to the amount that was present before the food was taken. In this connection it is interesting to notice that if the sixth and seventh dorsal areas are mainly affected, the pain will be increased almost immediately the food is taken into the stomach, and the patient may vomit practically unaltered food within twenty minutes of its ingestion. If, however,

the ninth dorsal area be mainly affected and the upper gastric areas are either absent, or but slightly developed, the pain will not be increased until a considerable time after the food has been taken, and vomiting may be completely absent.

The tongue remains unaltered, as a rule, in this form of gastric disturbance. If it were clean and red, or a little furred, soft and moist it does not alter, at any rate, at first, although the gastric symptoms are very marked. In the same way the action of the bowels and the character of the evacuations are not markedly altered, excepting in consequence of the rejection of food by vomiting, or some alteration in diet prescribed by the physician. Flatulence and the eructation of fluid into the mouth, are not usually symptoms of this condition.

Thus the whole of the symptoms are those of irritability of the stomach without obvious signs of gastric mischief. As soon as the outburst of fresh disease in the base of the lung clears up, or ceases to advance, the referred pain and superficial tenderness over the gastric areas ceases, or diminishes, and with this cessation or diminution of the pain and tenderness the gastric symptoms disappear. The explanation would seem to be that the outbreak in the lung pours pain impulses into those segments of the nervous system which stand in nervous connection with the affected part of the lung. But the stomach also stands in connection with the same segments, and thus becomes hyper-æsthetic, in exactly the same way as the testicle may become hyper-æsthetic owing to a renal calculus.¹

The first case I have chosen as an illustration shows the outburst of referred pain and tenderness, with the implication of the left base. The base of the lung was permanently damaged, but the referred pain and tenderness disappeared when the acute increase in the disease passed away.

The second case shows the disappearance of the gastric symptoms, with the clearing up of an acute outburst at the left base, and their reappearance when the base became again affected.

¹ Cf. BRAIN, 1893, p. 78.

Case 68.—To illustrate the second type of gastric disturbance associated with the invasion stage of phthisis. (*Sympathetic gastric hyper-æsthesia*). Edward O. (V.P.H., Dr. Thorowgood); aged 34, wire layer for electric light.

In the winter of 1890 had a bad cough and spat much phlegm. Got better, but continued to cough all through the summer. In the winter of 1891 got very bad again, and could not work. He attended Brompton Hospital, and much improved, but in October, 1892, became worse again. Wasted greatly, sweated at night; cough very troublesome. He again improved somewhat, but in October, 1893, got worse than ever.

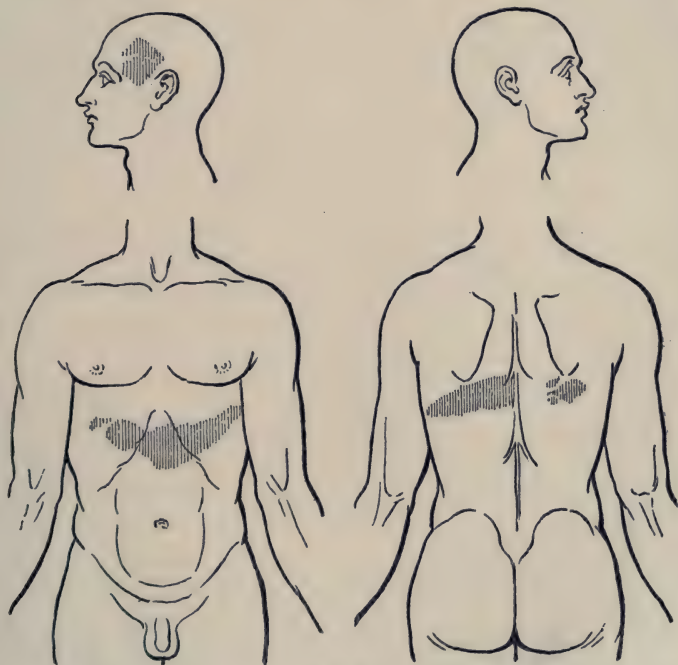


FIG. 21.

To show the areas of tenderness present in Case No. 68 during an attack of sympathetic gastric hyper-æsthesia.

On admission (April 9, 1894).—Small man of 34. Face, cachectic, of a somewhat greasy pallor. Slight pink congestive flush on the cheeks. No cyanosis. Considerably wasted.

Respiration 22. Accessory muscles of respiration acting slightly. Very short of breath, on exertion. Cough troublesome in the morning, but not troublesome in the daytime. Large amount of greenish-yellow, round masses of muco-purulent expectoration containing tubercle bacilli.

He complains of pain in the epigastrium, over the left costal margin, and below the angle of the left scapula. This pain is worse on the left side, than the right. Beautifully marked superficial tenderness. (Fig. 21). Headache over left temple accompanied by well-marked, superficial tenderness.

In Front: movement poor over both apices. Percussion note diminished over the upper three spaces on the left side. Over the left apex the breathing is bronchial, and bronchophony is heard. Over right apex breath sounds are weak, expiration prolonged, and vocal resonance diminished. Many crackling râles all over the front of the chest.

Behind: on the right side the note is dull, to the level of the angle of the scapula; over this area expiration is prolonged, vocal resonance increased and many rhonchi and râles are heard. From the angle of the right scapula to the base the breath sounds exceedingly weak, vocal resonance is much diminished and irregular, crackling sounds are heard. On the left side the percussion note is diminished to the level of the fifth spine. Over this area expiration is prolonged, vocal resonance increased, and many crackling râles and much inspiratory rhonchus is audible. From the level of the fifth spine downwards the breath sounds are harsh, expiration a little prolonged, but vocal resonance is unaltered. Many sharp râles and inspiratory rhonchi are heard over the lower part of the left lung.

Thus the signs in the lungs pointed to an old affection of the right base, and a recent affection of the left base.

Pulse 88, regular. Heart sounds normal.

Appetite very bad. He complains that the pain is increased by food. Feels sick, and retches within half-an-hour of taking food. Has vomited several times with this retching, apart from coughing. The vomit consists of almost unaltered food. Tongue small, moist, a little tooth indented. Bowels opened daily without drugs. Motions normal.

Liver and spleen not enlarged. Urine normal. Temperature rose every night to 100° F. (37.8° C.) falling in the morning to normal.

By April 20 all pain, headache and superficial tenderness had disappeared. He no longer felt sick after food. Food caused no pain. Appetite good.

The signs in the lungs on the right side remain the same. Over the upper part of the left lung behind, to the level of the fifth spine, the dulness is unaltered, and breath sounds and adventitious signs remain as before. Below this point all over the left base the breath sounds are good, vocal resonance normal, and absolutely no râles or rhonchi can be heard, or have been audible for several days.

On May 9. An exactly similar attack of nausea, vomiting, pain after food began, associated with another outburst at the left base. He left the hospital with persistent signs below the angle of the left scapula.

Case 69.—George R. (V.P.H., Dr. Sainsbury); aged 39, cellarman.

He was quite well till 1891, when he had an attack which was called "Influenza." He remained in bed for two weeks, and for the next three weeks was troubled with cough and spitting. July, 1892, began to suffer with pain in the left side and cough returned. January, 1893, had another attack of "Influenza" with pain in the left shoulder. He coughed, sweated at night, and lost much flesh. The cough has continued ever since. April, 1893, began to spit blood and the expectoration continued to be blood-stained for two weeks.

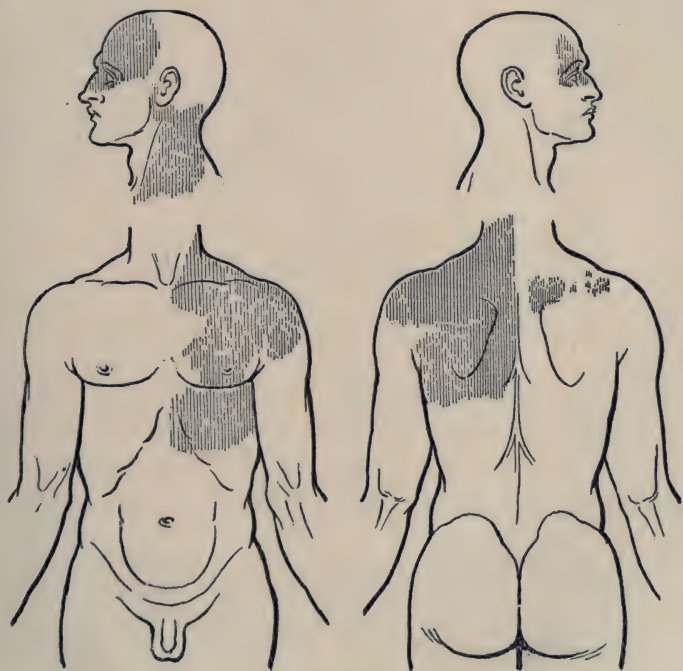


FIG. 22.

To show the areas of superficial tenderness in Case No. 69, produced by the invasion of the base of the left lung. This tenderness was associated with marked gastric disturbance.

Owing to the nature of his employment he takes alcohol somewhat in excess; five or six pints of beer daily.

On admission (October 16, 1893). Large well-built man, evidently somewhat wasted. Says he has lost 14 lbs. in six months. Face of a yellowish pallor. No flush in cheeks. No cyanosis. No profound anæmia.

Respiration 20. Accessory muscles quiet. Dyspnœa on exertion. Hard hacking cough. Spits up a considerable quantity of small muco-purulent yellowish masses tinged with bright or dark red blood.

Some pain in left supra-clavicular fossa, and over the posterior aspect of the left shoulder joint. Distinct superficial tenderness over the spots to which the pain is referred.

In front: right apex moves somewhat better than the left. Percussion note diminished in first two spaces on left side. Expiration is prolonged and vocal resonance increased as low as third space on left side. Fine dry râles heard over the same area. Over the right apex breath sounds are fair, vocal resonance a little increased, and a few scattered râles are heard.

Behind: on the left side the dulness extends to the level of the sixth spine. Expiration is prolonged and vocal resonance is increased over the area of diminished note. Crepitations are heard from the left apex to the level of the sixth spine, and when the left arm is placed on the right shoulder these crepitations are found to extend along the vertebral border of the left scapula. At the extreme right apex the percussion note is diminished, expiration prolonged, and a few crepitations are heard.

Pulse 80 regular. Heart sounds good. No murmur.

Tongue moist, a little flabby. Appetite excellent. Bowels opened daily. No vomiting or pain after food.

The temperature did not rise above 99° F. (37·2° C.).

November 17.—He complained of marked increase in intensity and extent of the pain on the left side of the chest. Wide-spread superficial tenderness. Headache and scalp tenderness.

The cough is more troublesome, expectoration no longer contains blood.

The right side of the chest remains as before, but there is a marked outburst of râles below the angle of the left scapula and in the left axilla.

Has lost his appetite. Feels sick after food. No diarrhœa.

November 26. Much pain on left side of the chest with superficial tenderness over the areas in the figure. Headache more marked on left side than right, and accompanied by marked scalp tenderness. (Fig. 22).

His appetite has completely gone. He feels sick and has vomited after food. The tongue remains clean. Bowels opened daily. Motions show no marked alteration.

The crepitations over the left base and in the left axilla have considerably increased since November 17.

Temperature reached 99·8° F. (37·7° C.) every night.

By December 20 all referred pain, superficial tenderness and headache had disappeared. He was taking his food well, and the nausea and vomiting were absent.

The left side of the chest behind was dull from the apex to the base. Over the upper part of the left lung behind expiration was prolonged and vocal resonance much increased, but below the

angle of the left scapula the breath sounds were very feeble, and vocal resonance was diminished. A few scattered râles were heard over the left base. No active signs in right lung. Heart's apex beat was normal.

Thus, the dyspeptic symptoms in this case came on with the implication of the left base, and disappeared when the base of the left lung became permanently affected.

(C) *True Gastric Dyspepsia.*

In the third type of gastric disturbance met with during the course of the invasion of the lung by phthisis, the referred pain and superficial tenderness bear no relation to the condition of the physical signs in the lungs. This is a true dyspepsia of gastric origin. The pain and tenderness extend over the sixth, seventh, eighth and ninth dorsal areas to a greater or less extent, but there is a marked tendency to the simultaneous development of referred pain and tenderness within the fourth cervical area. Now, this association of tenderness over the gastric areas of the trunk, with the areas about the clavicle and shoulder joint, is a common feature of true gastric disorders.

Pain after food with occasional vomiting, especially in the morning (apart from coughing), flatus, and the regurgitation of fluid into the mouth, are symptoms of this form of dyspepsia. The tongue is usually more or less furred, frequently with a red tip, sometimes it is soft and tooth indented, the bowels are always disturbed, constipation alternating with attacks of diarrhoea, the evacuations are frequently made up of small hard lumps of dark fæces with some leathery curds and darkish brown yellow fluid, and are distinctly offensive.

This form of dyspepsia is more directly amenable to treatment directed towards the gastro-intestinal tract, and a considerable number of the cases are improved by the ordinary rhubarb and soda mixture accompanied by the occasional use of cascara sagrada.

Thus, the distribution of the referred pain and tenderness, the condition of the tongue and evacuations, the flatus and regurgitation of fluid into the mouth, and the results of

treatment, all point to some disturbance of the stomach itself as the cause of this type of dyspepsia in phthisis.

Case 70.—Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis. Bernard McD. (V.P.H., Dr. Harris); aged 21, cook.

In May, 1892, he was serving in the Marine Artillery, when he broke his ankle. For this he was invalided, and he spent the

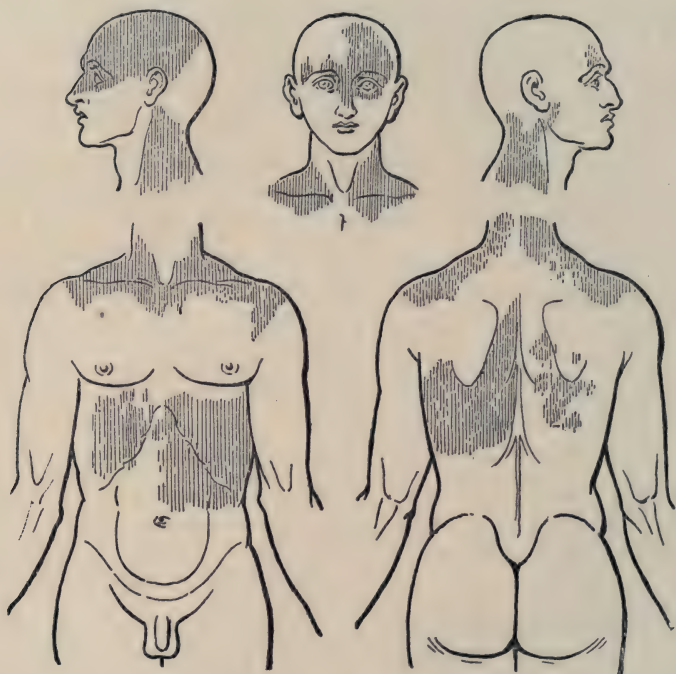


FIG. 23.

To show the areas of superficial tenderness accompanying an attack of dyspepsia, of true gastric origin, arising in the course of a case of phthisis [No. 70]

next five months nursing a friend, who died of "consumption of the lungs and throat." He then began to feel languid and faint, and "caught cold after cold."

August, 1893.—Cough came on badly; he began to sweat at night, and lost 7 lbs (3.4 kgm.) in weight.

February, 1894.—Hæmoptysis. Continued to bring up blood in small quantities for weeks.

Family History.—He has been entirely separated from his family for four years. One sister (who is in a convent) has had repeated hæmorrhage “from the lungs,” and is dying of “consumption.”

Admitted to V.P.H., July, 1894.

He suffered much from pain, vomiting, and alternating constipation and diarrhœa; coming on in attacks at varying intervals. The condition on September 7 was typical of several attacks which were watched during his stay in hospital.

September 7, 1894.—He is a well-built man of 5 feet 10 inches. Face thin; and there is much wasting about the neck. He has lost 3 stone (over 20 kgm.) in twenty months. No marked sweating at night. Face pale, and of a uniform earthy white. No flush. Lips pale, but distinctly blue. Temperature ranges to 101° F. (38·3° C.) at night, but sinks to 98° F. (36·7° C.) in the morning.

He has been somewhat freer of gastric disturbances until today. Tongue somewhat blue-red, moist; over the posterior part of the dorsum there is a triangular area of brown fur. Just before dinner he has a feeling of hunger and emptiness. Then he feels sick, and occasionally vomits. An hour after food, this same sinking feeling comes on again. He is passing green, very offensive, motions, three or four times in the twenty-four hours.

He complains of much pain, particularly in the left side, both back and front, from about the level of the sixth rib to the level of the umbilicus. It is most marked in the epigastrium, and behind the angle of the scapula. He also has pain in the posterior aspect of both shoulder joints, running up the back of the neck, and causing a feeling as if his neck was stiff. The pain is sharp, and leaves behind it a kind of “benumbed, painful feeling.” Then, when he moves, the sharp pain comes on again within the “benumbed areas.”

Headache over the forehead and temples. Hair is sore when brushed.

Marked superficial tenderness of body and scalp, as in fig. 23.

This pain is always more or less present now. It is worse before food; is somewhat relieved by food; and is worse again about an hour after a meal. Some flatulence; and the pain is easier when he brings up the wind.

Respiration 20. Sterno-mastoids acting. Very short of breath on exertion. Cough in occasional paroxysms; especially in the morning, when the cough causes vomiting. Expectoration in irregular, greenish muco-purulent masses, but not very profuse. (It has been tinged with blood since admission).

The left side of the chest is now dull from top to bottom, both back and front. Expiration is prolonged, and vocal resonance increased all down the left side, and in left axilla; and many crackling râles are heard. (The left base became affected on August 18, and has remained in the same condition ever since).

Over the right side of the chest the note is impaired as low as

the fourth rib in front, and the fifth spine behind. Over this dull area the breath sounds are bronchial. Vocal resonance is increased, and numerous fine crackling râles are heard. (The right side only was affected on admission in July.) The right base around and behind the angle of the scapula, and the lower part of the right axilla, are clear.

Palpitation and feeling of faintness on exertion. Pulse 88; small, regular. No abnormal sounds in the heart.

Urine, specific gravity 1022, acid, and with some albumen.

He died at the beginning of November, 1894.

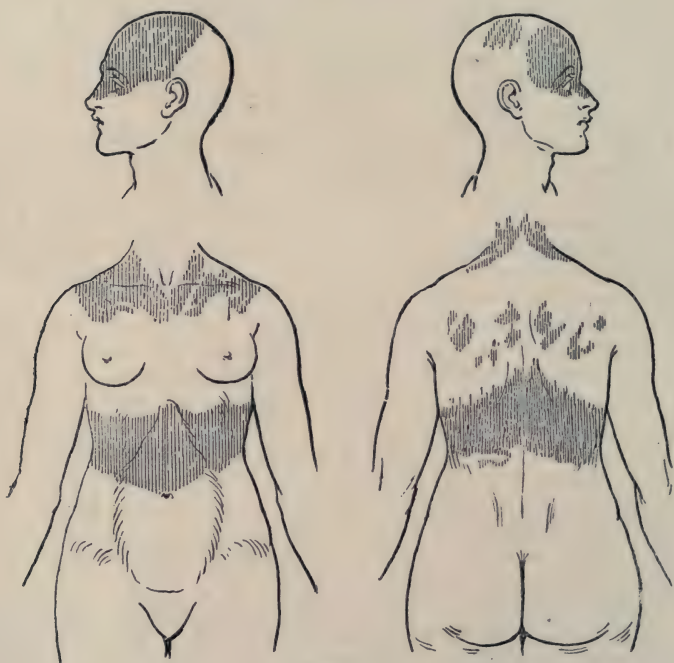


FIG. 24.

To show the areas of superficial tenderness accompanying an attack of dyspepsia of true gastric origin, arising in the course of a case of phthisis [No. 71].

Case 71.—Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis.—Minnie E. (V.P.H., Dr. Harris); aged 23, waistcoat maker.

In the winter of 1888-89 she began to have a dry, harsh cough, which passed off with the coming of the spring.

October, 1889.—She began to suffer from pain after food, and much vomiting. With this attack a hard cough came on again.

She was admitted to a general hospital, and there told she had a gastric ulcer. (Her abdomen still shows the scars of the frequent blisters that were then applied to the epigastrium.) She was well in ten weeks.

June, 1890.—The cough came on again, with nausea and pains in the limbs. Said to be influenza. In five weeks she was better, but very weak. August, 1890, a second similar attack, from which she did not recover for a long time.

Every winter the cough reappeared, but cleared off with the summer, until 1893. The cough then became constant.

September, 1893.—She began to waste rapidly, and to sweat profusely at night. The cough became much looser.

February, 1894. On admission.—Very marked purple flush upon the cheeks. Marked wasting. Sweats at night.

Respiration 35 to 40. Sterno-mastoids acting vigorously. Much subjective dyspnoea. Single troublesome cough. Brings up a large quantity of stringy aerated mucus.

Much pain at epigastrium and under shoulders, especially on the left side. Marked superficial tenderness over the sixth and seventh dorsal areas, with spotty tenderness over the fourth cervical area, on the left side. Widespread fronto-temporal headache, with superficial tenderness over the frontal mid-orbital, fronto-temporal, and temporal areas of the scalp.

Right apex moves better than the left. Percussion note diminished over left front. Over the upper two spaces on left side, in front, breath sounds are cavernous; bronchophony is audible, and many crackling râles are heard as low as the fifth intercostal space. Over the first two spaces on the right side the breath sounds are bronchial, and bronchophony is present, and numerous crackling râles are heard.

Behind: on left side, diminished note as low as level of fourth spine; breath sounds cavernous, with whispered bronchophony. Crackling râles from apex to around angle of scapula, and in the axilla. Over the right side: note diminished, as low as sixth spine. Over upper lobe: bronchial breathing and bronchophony; but opposite the level of third and fourth spines, an area of profound cavernous breathing and whispered pectoriloquy.

Pulse 100; small, regular. Apex beat, fifth space, internal to nipple line. Cardiac dulness slightly diminished. No abnormal sounds.

Tongue somewhat dark red, and not well moistened. Appetite poor. Bowels opened two to three times daily.

Liver and spleen not felt. Nothing abnormal felt in the abdomen.

Temperature sometimes touched 103° F. (39.4° C.) at night, and did not fall below 100° in the morning.

The signs on the left side became quieter, but the right side progressed rapidly. The pain and superficial tenderness became more marked on the right side.

At the end of March the gastric symptoms became a much

more prominent feature, and the superficial tenderness was very marked.

April 30.—She is vomiting green watery fluid in the morning, apart from cough, and before breakfast. The tongue is dry and red, with some irregular whitish fur, but not heavily coated. Appetite is very bad. She has been constipated, but the motions are now three or four in the twenty-four hours. The evacuations consist of some irregular brownish lumps, with much brown offensive liquid material.

She is complaining of much pain in the epigastrium and under the shoulder blades. She also has pain over the neck on both sides and over the shoulder joints. Very marked superficial tenderness, as in fig. 24. Headache over the temples and forehead, with superficial tenderness, as in fig. 24.

Liver and spleen not enlarged. Nothing abnormal felt in the abdomen. No marked signs of dilatation of the stomach.

The signs in the lungs have advanced rapidly. *In front*, there is dulness, with cavernous breathing and whispered pectoriloquy, as low as the fourth rib on the left side. On the right side, hollow breath sounds and whispered bronchophony, as low as third rib. *Behind*: on left side there is dulness to level of sixth spine, with cavernous breathing and whispered pectoriloquy to level of third spine. On the right side, dulness, with cavernous breathing and whispered pectoriloquy, as low as angle of left scapula.

The temperature does not vary more than about a degree, and the mean temperature is 101° F. (38·3° C.).

This case illustrates the true gastric disturbance of phthisis, and shows the association of the cervical areas of tenderness with those of the epigastrium.

It is frequently very difficult, in practice, to decide in which of these three groups to class some particular gastric disturbance that occurs during the course of a case of chronic phthisis. From a clinical point of view, such a division into three forms of dyspepsia would be scarcely necessary. But, from the physiological aspect, such a division is absolutely essential; for the three types are the expression of, at any rate, two fundamentally different processes.

Moreover, if we remember that two of these forms of dyspepsia are produced reflexly, whilst one is of gastrointestinal origin, we can understand the following facts, upon which most observers are agreed. It would seem that in spite of the notorious proneness of phthisical patients in this stage of the disease to suffer from gastric disturbance,

in the large majority of cases no abnormal activity, either of the mucous membrane or of the muscular walls of the stomach, can be discovered. On the other hand, certain cases show distinct secretory and motor abnormalities.

Again, it helps us to understand why, in a large proportion of the cases, the dyspepsia tends to disappear, or to alter in type, as the destruction of the lung progresses. For the two purely nervous forms, due to the destruction of the bases of the lungs, must cease when the changes in the lung assume a form incapable of generating referred pain. Thus the "reflex dyspepsia" is a very prominent feature of the invasion stage of the disease, when organic changes in the walls and coats of the stomach are uncommon; whilst it may be entirely absent in the third stage, when alterations in the walls of the stomach are of much more frequent occurrence.

This assumption of a reflex origin for many of the dyspepsias, which appear during the invasion stage of phthisis, helps us to understand why gastric symptoms are not a marked feature of pneumonia, pleurisy, or of those cases of phthisis where the destruction of the lung is mainly by cavitation. For complete consolidation prevents the appearance of referred pain, by throwing the nerve endings of the diseased portion of the lung out of play. Cavitation destroys the nerve endings in the lung, and is, therefore, unaccompanied by referred pain. Pleurisy causes a local pain, in which the splanchnic system of afferent nerves is not implicated. Thus we should no more expect reflex dyspeptic symptoms to accompany a pure pleurisy than we should expect them to accompany an inflammation of the skin of the chest wall of similar extent.

To sum up the results to which we have arrived in this section:—

The gastric disturbances, which occur in the course of phthisis, may be divided, *clinically*, into three groups:—(1) The prodromal dyspepsia, appearing before there are any marked signs in the lungs. This dyspepsia is frequently accompanied by marked referred pain and superficial tenderness. It seems to be associated with the cachexia which not infrequently heralds the onset of the disease.

(2) The dyspepsia of invasion, also accompanied by referred pain and superficial tenderness.

(3) The dyspepsia of dissolution, accompanied by marked signs of organic change in the walls and mucous membrane of the stomach, but unaccompanied by referred pain and superficial tenderness.

Physiologically, the second of these clinical divisions can be again divided up into three types, according to the causes which give rise to it:—

(a) A pseudo-gastric disturbance, due to the nausea and vomiting produced by the referred temporal headache, which stands in definite relation to the referred pain produced by the implication of the base of the lung.

(b) Gastric hyper-æsthesia, which owes its origin to the fact that the bases of the lungs and the stomach are supplied from the same segments of the central nervous system. Thus an affection of the base of the lung causes a disturbance within the sixth to the ninth dorsal segments, and the stomach becomes hyper-æsthetic by sympathy, just as the testicle becomes tender in some cases of stone in the kidney.

(c) True dyspepsia of gastro-intestinal origin, having no relation to the nature of the phthisical process, or the distribution of the disease within the lung.

In conclusion, I have to offer my sincere thanks to the staff of the Victoria Park Hospital for Diseases of the Chest, for the extremely liberal manner in which they have permitted me to make use of their cases, not only during the time I acted as house physician, but also during the last three years.

My thanks are also due to Mr. Lunn for permitting me to make use of some of the cases under his care at the St. Marylebone Infirmary.

ON THE INFLUENCE OF REAGENTS ON THE ELECTRICAL EXCITABILITY OF ISOLATED NERVE.

BY AUGUSTUS D. WALLER, M.D., F.R.S.

(Continued from p. 67.)

BEFORE proceeding to review the effects produced upon isolated nerve by reagents in solution, we must consider the conditions of observation. We are acting on the hypothesis that a current of injury between longitudinal and transverse points of the nerve depends upon greater chemical activity at the transverse, or more injured point, and that a current of action, or negative variation of the injury current, depends upon a superadded chemical activity most pronounced at the longitudinal, or less injured part. We shall therefore apply our reagent to this less injured part, and watch for an alteration in the rhythmical series of deflections. Before we know the separate action of water, alcohol, salt, &c., it is obvious that we may not proceed to apply solutions to the nerve. Looking back to the result of *e.g.*, chloroform vapour, we may assume an increased deflection and a diminished deflection, as signs of excitation and of depression respectively. But the possibilities of chemical reaction between an added fluid and the clay electrode itself need to be remembered, although this is no very formidable danger, for we shall not often be obliged to run into it; alterations of the galvanometer zero thus produced do not interfere with our observations of the deflection itself, and after all we can easily test some extreme instances. Nor will the alteration of resistance due to adding fluid to an electrode cause us much trouble, for we can at any moment gauge such alteration by closure of a key to send from the compensator a small standard current into the experimental circuit.

At first this mode of trial was promising of results. A drop of saline solution had no effect on the deflection, a drop of water diminished it but very gradually, a drop of muscarin solution abolished it, so did a crystal of common salt, a drop of chloroform, a drop of 10 per cent. alkali, a drop of 10 per cent. acid; in all cases the reagent being restricted to the longitudinal electrode and superjacent portion of nerve.

But a little further reflection makes plain to us the unworkability of this working hypothesis. It will not do to apply a drug in solution locally to the nerve on the longitudinal electrode. That electrode serves as the lead-off not merely to the portion of nerve lying upon it but also to adjacent portions. Such portions may or may not have been reached by the drug, may or may not have been affected by it. The conditions of observation are, in short, nothing like so favourable as in the case of a gas or other volatile drug, which, when passed into the gas-chamber, equally influences the whole nerve.

This reflection impresses upon us the need of submitting the whole nerve to the action of the drug. We may lift off the nerve, let it soak in a watch-glass of fluid to be tested, and replace it on the electrodes. This seems at first sight a clumsy proceeding. Or we may lay the nerve on the electrodes clinging to a strand of wool moistened by salt solution, then apply our fluid with a pipette to the, so to speak, imbedded nerve, to all parts of which the wool acts as carrier of fluid by virtue of capillarity. This, at first sight, seems neater, involves no disturbance of contact, and we may feel sure that the whole of the nerve has been submitted to the drug.

We must, of course, under these, as indeed under any circumstances, exercise discretion in the comparisons we make and the conclusions we draw. To compare variations before and after a solution has been added, or before and after a wet thread has been laid along the nerve, and to attribute the difference to an action of the drug, would be absurd, for the resistance has been altered and additional derivation paths have been established. What we may do,

though, is to learn with certainty whether an effect has been suddenly abolished or suddenly augmented, and failing sudden action, to witness the gradual development of a change dating from the moment of interference.

And—provided all reserve be made in the adoption of conclusions—we shall, in case it be required to ring the changes in a succession of trials, prefer the simpler and easier method of taking off the nerve, soaking it for a definite time in the test solution, and replacing it on the electrodes as nearly as possible in its original position. It is, in fact, better to deliberately remove and replace the nerve, passing it with a glass rod through watch glasses of reagent and of saline, than to attempt to put a moist thread on and off, for the nerve is pretty sure to cling, and if more than one trial is to be made the electrodes must obviously become contaminated.

A precaution of principal importance is to test the resistance after each re-adjustment; it is evident that failing this precaution, we cannot be certain of the significance of an increased or diminished variation which might be due to a diminished or increased resistance, chiefly of the nerve itself, according to the fluid employed.

We shall attend principally to the rhythmic excitation effects, laying little stress upon any shifting of the zero that may have taken place. As a matter of fact, however, it is surprising, when we consider the delicate adjustment of the spot, how nearly its original position is recovered if a nerve is taken off, washed in salt solution, and replaced as nearly as possible *in statu quo*. Nor is there usually any great alteration of resistance, which implies that the variation before and after the bath are not on any very different scale, as is the case with the thread device.

Within what limits it may be safe to draw conclusions as to the action of a drug, will be best illustrated by an example.

The first drugs that I happened to use, not because I had much reason to imagine them to be active upon nerve, but partly because they were the drugs we tried together on the heart, partly in pursuance of the idea that muscarin is a

general protoplasm poison, and that nerve is a very simple sort of protoplasm, were muscarin and its antagonist, atropine. I took muscarin as being a drug with which I was most familiar as regards its action on the heart, where, as you may remember, we saw reason to believe that it is more correct to regard muscarin as depressant of protoplasm rather than as a specific excitant of inhibitory ganglia. And it was with this drug and with the same solution that you saw used in the series of heart experiments, that I went through certain doubts and difficulties which we may find it instructive to review. I will summarise them as quickly as possible by running through the records of their occurrence.

Plate 206¹ was my first trial of the possible action of muscarin on nerve. A drop of muscarin solution was placed upon the nerve where it rested upon the longitudinal electrode. Here is the essential part of the protocol, the plate exhibits an unmistakable effect of the drug.

Time.				Deflection.
0 mins.	before muscarin 16 (regular)
2	„ after muscarin 12
3	„ 8
4	„ 5
5	„ 3
7	„ 1
10	„ ?
15	„ 0
30	„ 0

This looked all right, but on the next trials (plate 210) no marked effect, if anything an increased deflection.

Before muscarin	15 (regular)
After muscarin	19 (regular)
After more muscarin	17 (regular)

¹ Many of the plates used in this lecture have not been reproduced here. Measurements only, or curves summarising several measurements are given of the chief significant experiments, with the exception of figs. 2, 3 and 4, which are records of the three most common bromides, and of figs. 9 and 10, in which an active and an inactive reagent (aconitine and aconine) are contrasted. These examples will suffice for the present to justify the application of the method to drugs in solution. Other records, inclusive of some that are referred to in this lecture, will be given in the next number of BRAIN.

Plate 211.—No marked effect.

Before muscarin	23
After muscarin	25 (falling to 22)
After more muscarin	17 (regular)

Plate 212.—Doubtful effect.

Before muscarin, 15.

After muscarin, 15 falling steadily to 8 during the next 25 minutes.

Plate 213.—Still more doubtful effect.

Before muscarin, 16 (regular).

After muscarin, 22, falling steadily to 17 during the next 20 minutes.

This was growing monotonous, these negative results were failures, which for the moment I could not account for. They were failures because the positive result of the first experiment seemed so clear and non-accidental (barring the one hardly probable accident of a contaminated glass rod used to put on the muscarin) that one could not admit the inactivity or the doubtful activity of muscarin. One might, perhaps, feel tempted to fall back upon some refuge formula for one's ignorance and say, as has been said in a similar case of doubtful results, "instability of nerve," but unstable is precisely what a nerve is not, and the use of such a formula concerning experimental results is a very thinly disguised and pretentious way of confessing ignorance.

But anyhow, the "failures" gave me pause, and led me to make reflections as to the correctness of strictly local application at a longitudinal electrode, and to try more extensive application of the drug by the soaked thread, or by actually bathing the nerve in a watch-glass. This cleared the matter up, and showed that the more extensive application of the drug was the proper mode of testing. I think, but of course cannot be sure, that the first positive and subsequent four negative results were owing to the fact that in the first trial, not knowing whether anything was to happen, I made very sure that the nerve should have its muscarin solution, and daubed it on freely, while in the next four trials, expecting an elegant effect, I applied a mere droplet with great care and neatness to the very centre of the nerve lying across the longitudinal electrode.

Forgive me for dwelling on such a trivial matter, it was a by no means unimportant trifle, inasmuch as the momentary difficulty helped to point out what I believe to be the right way of trying a nerve by a drug, and it is not an un instructive illustration of a stumbling-block becoming a stepping-stone.

Plates 219-220.

Time.						Neg. Var.	Gradn. 0.0002 volt.
0 mins.	Nerve only	—	11
1	„	42	—
4	„	42	—
5	„	Nerve + saline thread	—	55
6	„	13	—
7	„	13	—
9	„	13	—
12	„	13	—
14	„	Nerve + muscarin thread	—	60
15	„	7	—
20	„	5	—
25	„	3	—
30	„	0	—
31	„	Nerve + atropin thread	—	60
35	„	0	—
65	„	0	—
95	„	0	—

Plates 221-2-3.—Muscarin then atropin.

Time.					Deflection.	Gradn. 0.0002.
0 mins.	Nerve only	15 (regular)	10
5	„	Nerve + saline thread	8 (regular)	—
10	„	Nerve only	20 (regular)	—
15	„	Nerve + muscarin thread	—	20
16	„	9	
17	„	7.5	
18	„	6.5	
19	„	5	
20	„	3	
21	„	2	
22	„	1.5	
23	„	1	
24	„	trace	
30	„	Nerve + atropin thread	0	20
60	„	0	
90	„	0	

You naturally inquire next about atropin. So did I, and in the last two experiments with a negative result. This is

unsatisfactory. If the muscarin effect was a true physiological modification we should, from the analogy of cardiac muscle, expect that modification to be antagonised by atropin. But I must pass on, leaving this unsatisfactory result as it stands for the present¹; there are other and far more satisfactory results that demand our attention, results which, however, were the immediate outcome of this check. And when you have realised what a rich harvest lies around us, I think you will forgive me for not remaining behind to gnaw this very tough and in itself probably not very nutritious root.

Once satisfied of the ease, simplicity, and clearness with which the action of a solution upon nerve can be tested we have but to go and take groups of substances as fast as we can think them out. I have already too much gathered material to be able to lay it before you in detail, but must, on the contrary, pick, and choose, and concentrate.

To that end I will put before you—

(1) Data relating to the haloid salts, with special reference to the bromides in particular.

(2) Data relating to some alkaloids and other hypnotics, taken almost at random.

(3) Data relating to antagonism.

Here are nine experiments showing the action of halides, all the experiments having been made under—as far as possible—constant conditions. In each case the nerve was bathed for one minute in a solution of decimolecular strength. Judge for yourselves whether the depressant action depends *more* upon the basic or upon the acid moiety,—in KBr, *e.g.*, whether the K or the Br is the predominant partner. And take note that on these data you can only judge whether one or other moiety has a predominant action; other data would be necessary before you could tell what (if any) is the share of the subordinate moiety in the total action. You may, indeed, make a shrewd guess at the facts of the case, from my use of the words “subordinate” and “predominant.” And here is a

¹ I have not yet found time to repeat these experiments, and am still doubtful whether, or no, muscarin and atropin affect nerve fibres. In any case, their action (if any) is much less marked than that of *e.g.*, aconitin, and cocain and physostigmin.—(July, 1896.)

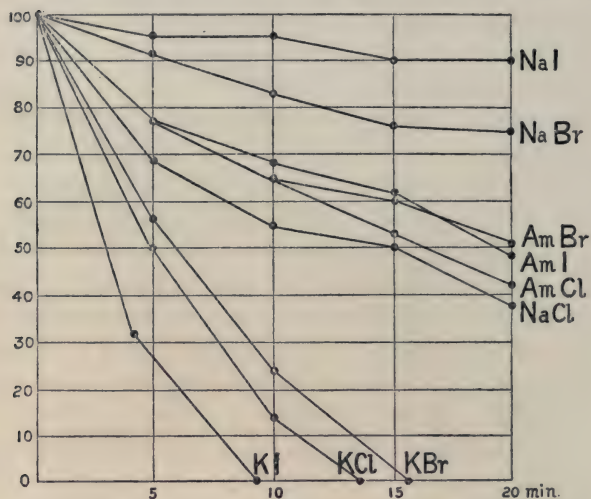


FIG. 1.

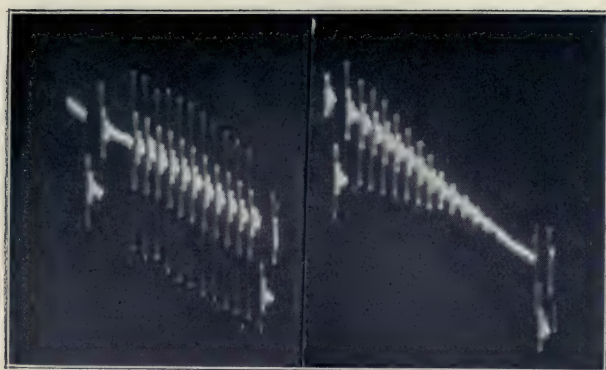
Curves exhibiting the effects of the chlorides, bromides and iodides of ammonium, sodium and potassium, upon the electrical excitability of isolated nerve.

In each instance the nerve, after its normal response had been recorded, was bathed for one minute in a decimolecular solution of the salt, and the response recorded for a further 20 minutes.

To construct the diagram, each of the nine series of measured records was reduced to a common measure, the normal response being taken as 100. The actual electromotive value differed in the different experiments of this figure, but may be regarded as approximately 0.001 volt.

CHLORIDES.				BROMIDES.			IODIDES.		
Plate No.	(281)	(282)	(280)	(274)	(272)	(271)	(283)	(286)	(285)
Normal	100	100	100	100	100	100	100	100	100
	AmCl	NaCl	KCl	AmBr	NaBr	KBr	AmI	NaI	KI
5 min.	76	68	50	75	91	55	75	95	32
10 "	65	55	12	65	82	22	68	95	0
15 "	53	50	0	60	75	0	61	90	0
20 "	41	37	0	50	75	0	48	90	0
25 "	30	27	0	—	—	—	—	—	—

FIG. 2.

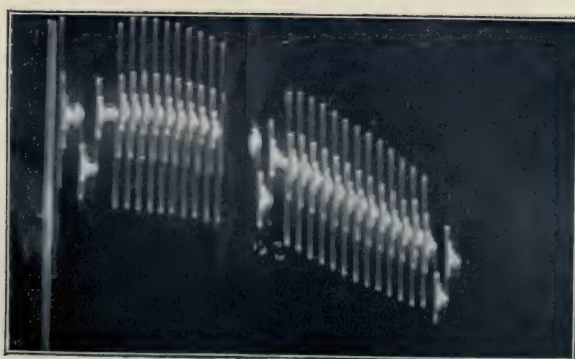


Before.

↑
KBr. $m/10$.

After.

FIG. 3.

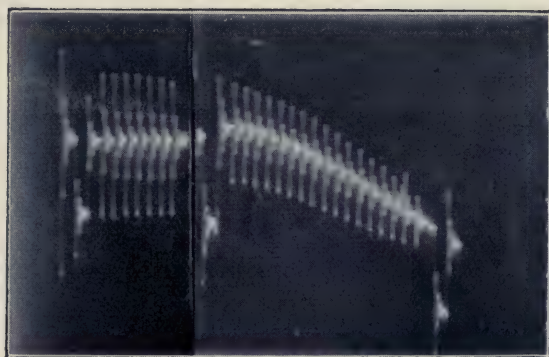


Before.

↑
NaBr. $m/10$.

After.

FIG. 4.



Before. ↑ After.
 AmBr. $m/10$.

FIGS. 2, 3 AND 4.

Influence upon nerve of the bromides of potassium, sodium and ammonium.

In each case the nerve, after its normal response has been recorded for about 10 minutes, is bathed for 1 minute in a decimolecular solution of the bromide, and the response recorded for a further period of about 15 minutes.

The bromide of potassium produces most effect, that of sodium least effect.

single plate (No. 520), exhibiting the action of strontium bromide, which will suffice to prove that the basic moiety is not the sole efficient partner nor even always the predominant partner. NaCl , NH_4Cl , KCl are, as you remember, depressant salts; SrCl_2 is anti-depressant; K and Sr in the respective chlorides are the principal and opponent elements, Sr being, indeed, the most prominently anti-depressant element that has yet come under my notice in these experiments. Yet the compound SrBr_2 is, as you see, a depressant salt. As we shall see in a moment, the chlorides of strontium and of potassium are mutually antagonistic; the bromides of strontium and of potassium are not so.

The predominant influence of the basic moiety—in the present case the greater depressant action of potassium than of sodium—is illustrated in other instances; thus to name only such cases as I have experimentally verified, caustic potash is more active upon nerve than caustic soda, potassium carbonate than sodium carbonate, potassium cyanide than sodium cyanide, taken, of course, on an equi-molecular scale of solution.

On the other hand, the subordinate influence of the acidic moiety—not very marked in the series of salts we have just reviewed—is clearly seen in the case of the series EtCl , EtBr , EtI . There is a general resemblance in the action of these three compounds, they are anæsthetics of the ether type, and have each, at one time or another, been recommended as such in actual practice. But their effects are by no means identical: ethyl bromide is apt to be irritant; ethyl chloride gives an anæsthetic effect that is apt to be more fugitive than desired; ethyl iodide is apt to produce a lingering after-depression. These are, I believe, the kind of character that the three drugs have earned clinically, and volatility is no doubt a factor in the result, thus ethyl chloride has found its most extended practical application as a local anæsthetic in dentistry, on account of its volatility. But the point to which I am now inviting your attention is that the general action of ethyl varies with its acidic companionship. Fig. 2 is a summary of actual experiments, of which the measurements are

given below; and you will, perhaps, be inclined to recognise in the results there embodied indications towards the selection of anæsthetics that, in this case, at least, are in unison with the slowly acquired conclusions of clinical experience—I had nearly said experiment. Imagine, for a moment, that clinically untried drugs are before you—ethyl dioxide, ethyl chloride, ethyl bromide, and ethyl iodide, you would in all certainty, if you used these nerve experiments as your sign-post, follow the way that has been followed by practical anæsthetists, rejecting the bromide and the iodide, and adopting almost exclusively the ethyl dioxide as your ordinary anæsthetic.

The conclusion to which I have been led by the study of the action of salts upon nerve may provisionally be summed up in this succinct formula.

A salt AB, taking effect upon living matter, acts in its compound capacity AB, not in an individual capacity A, nor B, nor $A + B$; in such compound action, B is more effective than A.

Or, figuratively expressed, the action of the firm composed of A and B is that of the firm AB (where A's action is modified by B's, and B's modified by A's, giving a total action expressible as AB's) not that of the partner A, nor that of B, nor that of A plus that of B. In the total action AB, the influence of B predominates.

The action of KBr is not that of K only, nor that of Br only, nor that of K plus that of Br; it is the action of KBr as a whole, but an action in which the characteristics of K are most distinctly recognisable. K is the predominant, though not solely, effective partner. KCl, KBr, KI, are not of identical effects, nor in the anæsthetic series are C_2H_5Cl , C_2H_5Br , and C_2H_5I ; but in the two series it is the basic element potassium and the basic radicle ethyl that characterise the two groups; all three members of the first are depressant; all three members of the second are anæsthetic.

Just one remark touching the bromides, which, as you well know, are prominent among the calmatives of the *materia medica* in correction of nerve exuberance, such as epileptic and other convulsions. The bromide of potassium

is *the* calmative bromide, but in cases where this bromide is found to be too depressing, those of sodium, or ammonium, or lithium, or calcium, or strontium, are prescribed in its place. I have tested all these bromides upon nerve; of all the effects are similar in character, though differing in degree; there is no augmentation by CaBr_2 , or by SrBr_2 . Evidently these bromides may be tolerated when KBr is found to be too depressing. But I must take some other occasion for discussing this bromide question in detail.

Here are records showing the antagonism between members of the sodium group and members of the calcium group—an antagonism that was first made known to us by Ringer's experiments with the frog's heart.

A few words of orientation are desirable here.

Physiological antagonism pure and simple should consist in this: an impulsion towards greater or smaller activity due to one set of molecules, neutralised by another set of molecules acting in the opposite sense. And an antagonism in its truest sense should be *simultaneous* antagonism, so to speak, a tug of war between two opposed sets of molecules acting simultaneously upon the protoplasm of contention.

As ordinarily studied, the antagonisms physiologically examined, such as muscarin-atropin, eserine-atropin, potassium-calcium, &c., have been *successive* antagonisms; the effect produced by a given reagent has been abolished by the subsequent administration of a second reagent.

Towards the formation of clear conceptions in pharmacodynamics, this distinction between simultaneous and successive antagonism should I think be made, and we need not stop to discuss the cases in which, owing to unequal rates of diffusion, two simultaneously applied reagents might successively come into effective action.

The distinction drawn between simultaneous and successive antagonism will help us to distinguish between what may be termed "true" and "false" antagonism. In a true successive antagonism, the physiological effect of one reagent is abolished by a second reagent of opposite effect, independently of any mere physical removal or chemical

neutralisation of the first reagent ; in a false antagonism, the first reagent is, so to speak, actually turned out by the second, or chemically neutralised, just as salt is washed out of a sponge by water, or an acid is neutralised by an alkali. Such effects should, I think, be excluded from our notion of physiological antagonism.

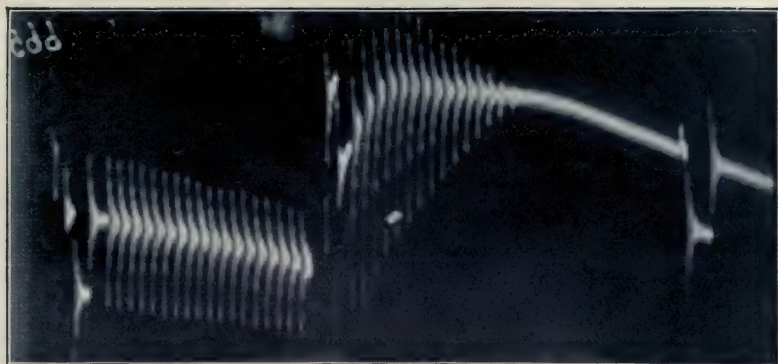
Here, then, are three cases on nerve which may serve to illustrate my remarks.

The first is a case of *false antagonism*. (Plate 256). In consequence of a one minute bath in a semi-molecular solution of NaCl (2.9 per cent.), the excitability of the nerve has declined. In consequence of a subsequent one minute bath in distilled water, the excitability of the nerve has been temporarily restored. We shall not, on that account, designate NaCl and H_2O as physiological antagonists. NaCl has been washed out by H_2O .

The next is a case of *successive antagonism*. In consequence of a one minute bath in a decimolecular solution of $SrCl_2$ the excitability of the nerve has been augmented. Half-an-hour later, with the strontium augmentation still present, the nerve was immersed for one minute in a decimolecular solution of KCl, in consequence of which excitability rapidly declines. This rapid decline is not a mere effect of removal of strontium ; under similar conditions, but with decimolecular NaCl in place of KCl, it did not take place. I may add that the Sr *versus* K antagonism is mutual ; this particular nerve was a little revived by a second strontium bath, and I have other records of a Sr increase after K decrease.

Here, finally, is a case of *simultaneous antagonism*, in which, after having found that dubious results ensued from immersion of the nerve in a mixture of equimolecular antagonists (equal volumes of decimolecular KCl and $CaCl_2$), I bathed a nerve for one minute (*a*) in a solution composed of six parts $CaCl_2$, and four parts KCl ; and (*b*) in a solution composed of four parts $CaCl_2$, and six parts KCl. In the first case Ca gets the best of it, and the series of effects is on the increase. In the second case K gets the best of it, and the series of effects is on the decrease.

FIG. 9.



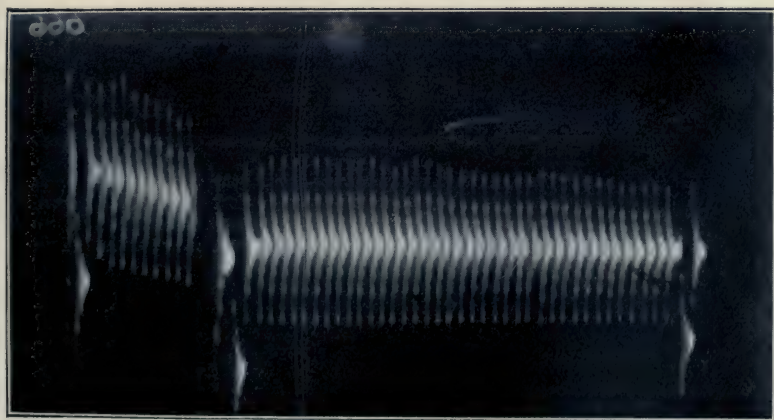
Before.



After.

Aconitine 0.33 %.

FIG. 10.



Before.



After.

Aconine 1 %.

Finally, let me again direct your attention to some alkaloids. We have already had something to say to muscarin and atropin; but as regards definiteness of result, the choice, or rather, the accident that led us to test these drugs first, was by no means encouraging; the result did not come out black and white, but a dirty grey. Further orientation experiments with other alkaloids have been far more satisfactory, yielding us results of the kind required in an early stage of investigation, definitely positive and definitely negative. Let me, however, at once guard the statements that I shall have to make as regards the negative action of a drug; they are comparative, and valid only under the conditions of time of immersion and strength of solution adopted in these experiments. We shall speak of a drug as having little or no action when its 1 per cent. solution has little or no effect upon a nerve immersed in it for a period of one minute, but such a statement must not be taken as committing us to a denial of any action whatever by the drug acting in stronger solution or for a longer period.

One of the best black and white contrasts that has presented itself has been between aconitine and aconine, the latter you may, for the moment, regard as being chemically a fragment of the former. Subject to what has just been stated, *i.e.*, under our conditions of experiment, aconitine promptly abolishes the excitability of nerve; aconine has no such effect. In order to bring out with any distinctness the gradually developing effect of aconitine, its solution must be taken below 1 per cent., at which strength the nerve is usually killed on the spot. Fig. 9, and its curve in fig. 6, exhibit the effect of a one-third per cent. solution of aconitine, *viz.*, the response of the nerve is at first exaggerated, subsequently abolished, somewhat as happens when the drug is applied to the excised heart. Fig. 10, and its curve in fig. 6, exhibit the comparative inertness of aconine.

These are chiefly orientation experiments, and I shall not enter into much detail at present, but shall simply bring before you a selection of such preliminary experiments, made with a view of ascertaining whether a given drug is to be characterised as active, or as inactive, in relation to the

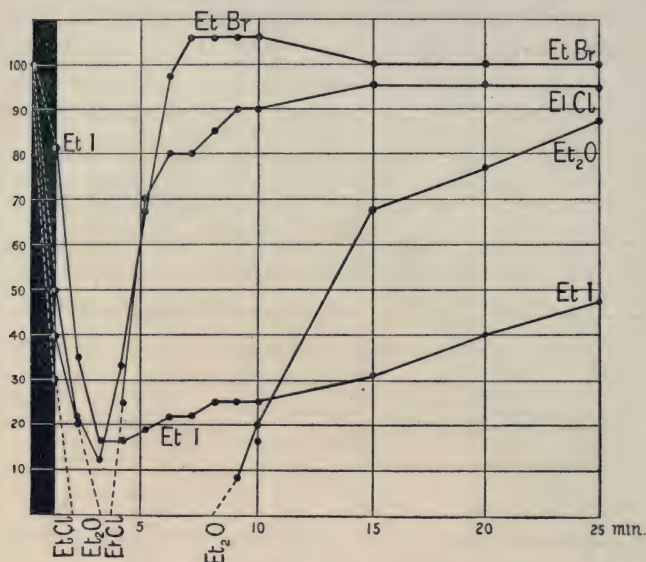


FIG. 5.

Curves exhibiting the anæsthetic effects upon nerve of ordinary ether (Et_2O), and of the chloride, bromide and iodide of ethyl (EtCl , EtBr , EtI).

In each case the nerve, after its normal response has been recorded for about 10 minutes, has undergone the influence of the anæsthetic vapour, blown through the nerve-chamber for 1 minute; the record has been continued for a further period of about 20 minutes.

TIME.				
0 min.	90		100	100
5 "	95		100	100
10 "	100		100	100
	Et_2O		EtCl	EtBr
1 min.	50		30	40
2 "	20		0	21
3 "	0		0	12
4 "	0		25	33
5 "	0		70	67
10 min.	20		90	106
15 "	68		95	100
20 "	77		95	100
25 "	88		95	100
30 "	95		95	100
			EtI	
				81
				34
				16
				16
				19
				25
				31
				40
				47
				52

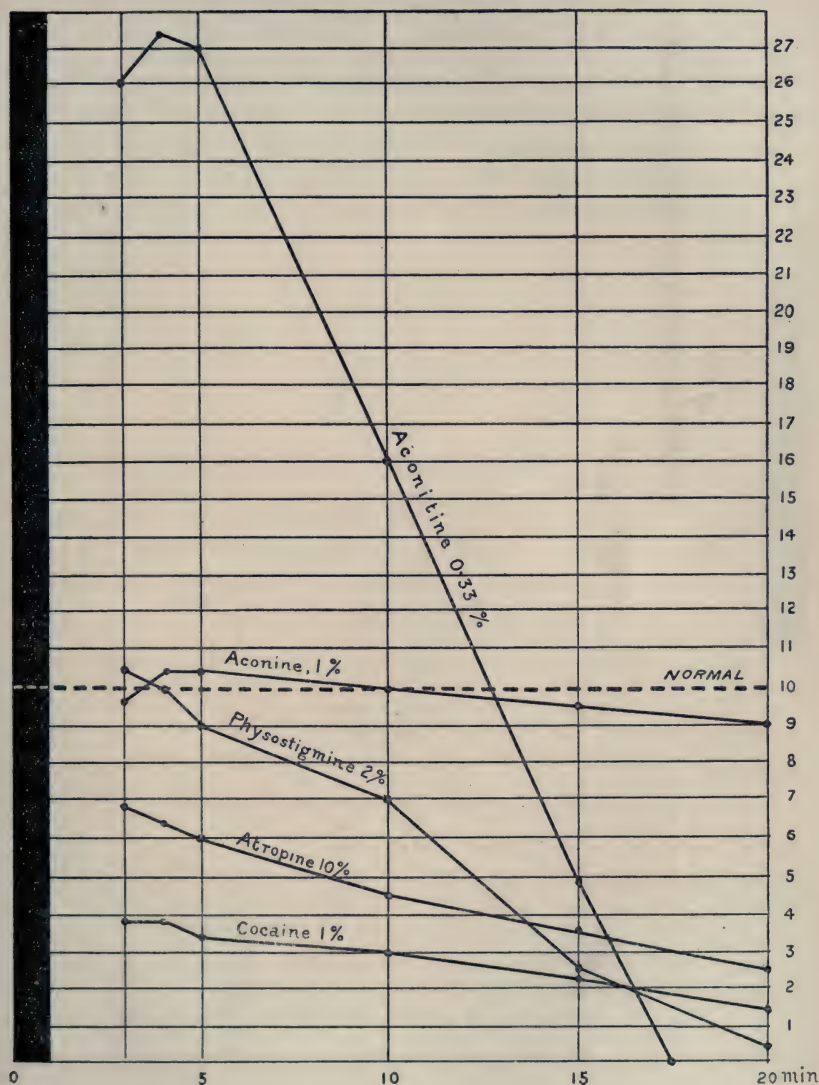
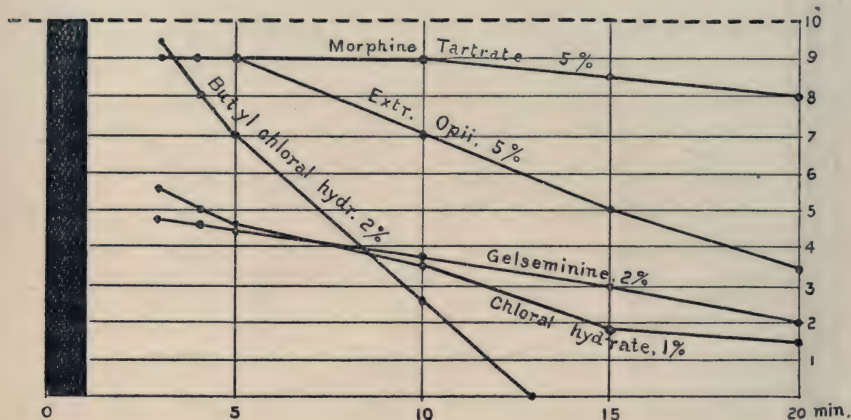


FIG. 6.

Curves exhibiting the effects upon nerve of:

(665) Aconitine hydrochloride	0.33 per cent. solution
(666) Aconine hydrochloride	1.00 " "
(355) Physostigmine sulphate	2.00 " "
(3,129) Atropine sulphate	10.00 " "
(840) Cocaine hydrochlorate	1.00 " "

electro-mobility of the nerve. The rule of procedure has been to take for about ten minutes, at intervals of one minute, the response of the undrugged nerve, then to immerse the nerve for one minute in a given solution, then to resume the observation on the now-drugged nerve for a further period of twenty to thirty minutes. The actual records are laid out for your inspection on that side-table; our review of the

FIG. 7.¹

Curves exhibiting the effects upon nerve of:

(567) Morphine tartrate	5 per cent. solution.
(724) Extract of opium	5 " "
(308) Gelseminine	2 " "
(321) Chloral hydrate	1 " "
(287) Butyl chloral hydrate	2 " "

results will be facilitated and accelerated by these diagrams (figs. 6, 7 and 8), in which the measurements taken from the records have been reduced to a common measure—the normal response of the undrugged nerves being taken as 100, and its altered response after drugging, indicated, therefore, by rising or falling percentage lines, time after drugging being

¹ In the experiments from which figs. 6, 7 and 8 have been constructed, the nerve, after its normal response had been taken for about 10 minutes, was removed from the electrodes, bathed in the solution named for 1 minute, and replaced on the electrodes. The deflections seen on the actual records at beginning and end of series of responses (figs. 2, 3, 4, 9 and 10) are standard deflections by an electromotive force of 0.001 volt. In the curves of figs. 5, 6, 7, and 8, and in their tabulated measurements, the reduction to a common measure (normal = 100) has been carried out as in the curves of fig. 1. But in order to indicate whether the modification of the nerve by a drug has been made on a series of increasing, decreasing, or constant magnitude, the normal point of departure = 100, has been taken at the end of the normal series of responses, just before the nerve has been drugged, and the measurements given of 5 and 10 minutes before this point: e.g., in fig. 9, there is a diminishing series before the nerve has been drugged, and the normal point = 100, has been taken at its end.

marked along the abscissa. With regard to these individual experiments, there is little more to be said than may be gathered on the face of the diagrams themselves, or of the records, if you care to examine them. There are one or two points, however, to which I may call your attention.

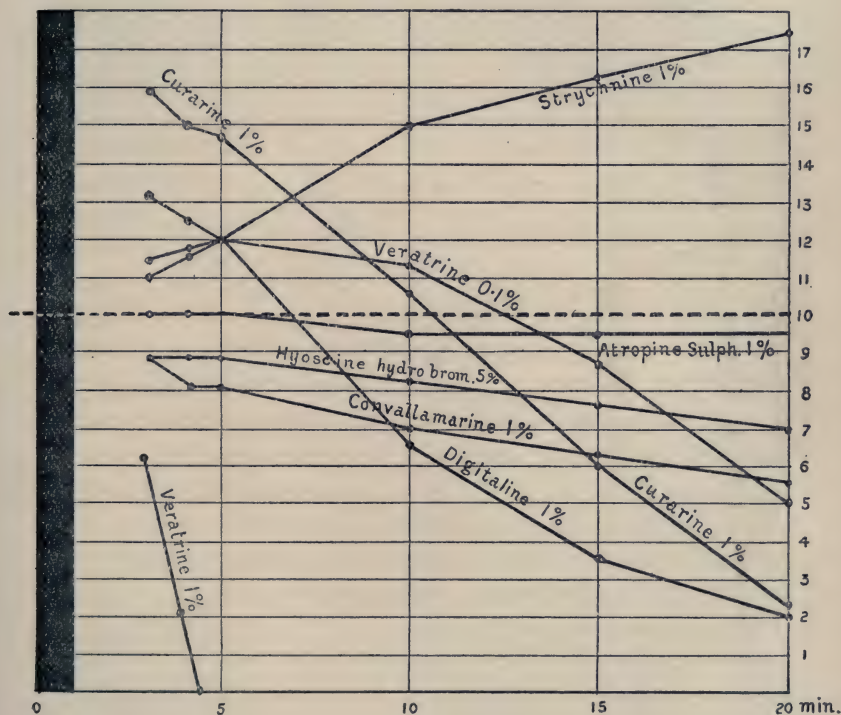


FIG. 8.

Curves exhibiting the effects upon nerve of:

(3,125)	Curarine	1.0	per cent. solution.
(3,128)	Strychnine sulphate	1.0	" "
(3,195)	Digitaline	1.0	" "
(3,196)	Convallamarine	1.0	" "
(3,201)	Hyoscine hydrobrom.	5.0	" "
(3,132)	Atropine sulphate	1.0	" "
(3,122)	Veratrine hydrochlor.	1.0	" "
(3,200)	Veratrine hydrochlor.	0.1	" "

Morphine (in this case in the form of tartrate, also I may add in that of the hydrochlorate; the bimeconate I have not yet examined) produces remarkably little effect. The observation used in the construction of the curve (I have

repeated it three or four times with a similar result) was made with a five per cent. solution of morphine tartrate.

It naturally occurs to one to ask whether the crude drug, from which morphine among other principles is obtained, is as inactive on nerve as morphine itself seems to be. Plate 724 (represented in the curve of fig. 7) gives the answer, and indicates as possible that some constituent of opium, other than morphine, is effective in the narcosis of nerve. The experiment has been repeated two or three times with similar results, but obviously requires development by other experiments.

Gelseminine, chloral hydrate, butyl chloral hydrate (which is clinically regarded as having a special predilection for the fifth nerve), and cocaine, tell their own story; but it is only with the last-named drug that I have made experiments in any number; and specimens of different source seem to act more or less forcibly, always however in the direction of depression. Atropine in 1 per cent. solution (fig. 8) has little or no action, in 10 per cent. solution (fig. 6) seems to have acted; but is it fair to put a drug in such an exaggerated concentration upon an isolated nerve, and then say that it acts upon nerve? I think not. Hyoscine (fig. 8), which is of the same family with atropine, is hardly less inert as regards isolated nerve, although clinically it is an undoubted and most potent sedative. One naturally reflects on possible differences of effect, according as a drug encounters grey cerebral matter by channel of the circulation, or medullated matter by direct application; some drugs are general anæsthetics through the brain, others are local anæsthetics at their seat of application; that is the empirical fact with which we must be content, and concerning which I know of no plausible explanation. Veratrine, like aconitine, is a local anæsthetic, and like it, is very active upon the nerve; in 1 per cent. solution the response is promptly abolished (see fig. 8); in 0.1 per cent. solution even, the response, after undergoing augmentation, suffers fairly rapid diminution.

Of all the curves plotted out in figs. 6, 7 and 8, the only one that exhibits prolonged and steady increase is that of strychnine; further experiments will, however, be required

before I shall venture to conclude that this is a regular effect. The observation is an isolated one, and the effect may therefore have been accidental.

Two other drugs that I have tested have given regular results, as indicated by the curves in fig. 8. Curarine—to a physiologist, one of the most interesting of drugs; to a chemist, not yet possessing any legal status—is distinctly active. Digitaline—clinically, of the greatest importance as a cardiac tonic; but chemically, figuring under many disguises—has likewise a distinct local action upon the nerve.

Convallamarine, allied to digitaline as regards its action upon the heart, has a similar but less pronounced action upon the nerve (in fig. 8).

These are, I am well aware, in relation to the magnitude of the subject, very fragmentary results. So far, however, from offering an apology for introducing the matter to you in such unfinished fashion, I am disposed to claim credit for so doing, inasmuch as contact with a growing surface of knowledge, physiological or otherwise, is the most valuable lesson that a teacher is able to offer.

You now know at least as much about the matter as I do; you have taken part in the preliminary difficulties of technique and in the avoidance of fallacies. As you know, I had no idea where we were going, and least of all any utilitarian anticipations, when I set up apparatus to show you a presumably delicate experiment. I used to think it a ticklish matter to exhibit without fail the negative variation of nerve; it has turned out to be easy. You have seen it going by the hour; you have also seen how easy it is to determine on nerve the quantitative ratio between the stimulus and the effect; and, finally, how satisfactory a test-object nerve affords as regards the unambiguous results of experimental interference. The apparatus (galvanometer, coil, rheostats, condenser, &c.) are, comparatively speaking, delicate apparatus, but by no means so delicate that they may not be “minded” by ordinarily skilled hands. Then I wanted to show you the *autographed* results of such observations, and you have seen how this was done with

the roughest possible apparatus—an old soap-box with a horizontal saw-cut; an ordinary photographic quarter-plate carrier fastened to a bent pin at the end of a thread, and let down by a wheel fastened to the minute axis of a common American clock. Nor was the gas-chamber a very complicated or expensive piece of apparatus. It would cut a very sorry figure in a modern laboratory or in an instrument maker's catalogue.

Yet a familiarity with such common things has not, I am certain, bred contempt in you for the questions that we have attacked together, but rather a curiosity for further knowledge, and a sense of its accessibility by simple means, and by "constantly thinking unto it."

Just consider what a vast field of knowledge lies open to us ready to be explored by this clean and keen little weapon—a tag of frog's nerve. I meant more than an evil-tasting joke in saying that a frog's nerve conducted itself like an ordinary man when subjected to carbon dioxide, to alcohol, and to tobacco smoke. I also had chloroform and ether in my mind, their glaring difference on nerve, the glaring difference in their death-rates when used as anæsthetics. Will you have ether or chloroform? Will you, as in America, use ether whenever you can, or as in England draw up death-rate statistics of chloroform, and appoint commissions to debate whether deaths by chloroform are by the heart or by the lungs? Then turn your thoughts to the pharmacodynamics of living matter; where may not these little tags of nerve lead us along its intricate paths? I do not know, but I feel myself entitled to repeat the exclamation used by my father in 1850, when he had found that the nerve fibre was kept good by its nerve cell—"A cloud as small as a man's hand."

Yet as observations are multiplied, and with them the number of exceptional or anomalous results, one may not resist a feeling of considerable perplexity. Question after question has unmasked itself from behind each new experiment; and one may well feel oppressed by the extent of possibilities before us, oftener than contented by the few scanty facts hurriedly surveyed.

I am not sure whether muscarine and atropine are to be put down as active or not on nerve-fibres. I only know that they have no activity comparable with that of cocaine or aconitine. I am not sure whether the antagonism between potassium and calcium is true or false. I have sometimes failed to obtain augmented action by strontium and by calcium, and I do not know what may be the conditions of the appearance or non-appearance of their effects.

Unfortunately, for the moment—yet no doubt fortunately in relation to the ultimate issue—the simple case of a nerve with a current of injury giving a negative variation when excited, is by no means simple, when its elementary explanation is pressed for. We have to study other cases of excitatory effects and after-effects, positive effects and positive after-effects, as well as negative effects of excitation, and the effects of reagents upon these. And then we shall find ourselves in a maze of possibilities, involving the study of electrotonic currents, of polarisation increments, and again, effects of reagents upon all these. We shall have to wade through many perplexities and complications before we reach to any degree of elementary simplicity, and, if I do not forbear exhibiting this damping outlook to you, it is because I wish you to put their proper value upon simple and easy ideas, and to realize that if they are easy when found, they are difficult to find and to make easy. I do not know where we may be going from this point, but the clues that I intend to pursue are certain suppositions relating to simultaneous negative and positive phenomena in nerve, connected with its polarisation and depolarisation, and belonging perhaps to the general doctrine of dissimilation and assimilation that we have already considered in connection with retinal phenomena. And the reagent, from the minute study of which we may feel most hopeful of further information, is carbon dioxide; it is the prominent dissociation product of living matter. We have seen that it profoundly modifies the excitability of living matter; the first part of our task will therefore be to study its effect upon the polarisation and depolarisation of medullated nerve.

TIME.	(No. 321).	(No. 287).	(No. 3,125).	(No. 3,128).
0 min.	100	100	94	100
5 "	100	100	97	100
10 "	100	100	100	100
	Chloral hydrate. 1 % for 1 min.	Butyl chloral hydrate. 2 % for 1 min.	Curarine. 1 % for 1 min.	Strychnine sulphate. 1 % for 1 min.
13 "	55	96	155	111
14 "	50	83	150	111
15 "	45	69	145	120
20 "	35	28	105	150
25 "	20	0	60	162
30 "	15	0	25	175

TIME.	(No. 3,195).	(No. 3,196).	(No. 3,201).	(No. 3,132).
0 min.	100	112	100	100
5 "	100	106	100	100
10 "	100	100	100	100
	Digitaline. 1 % for 1 min.	Convallamarine. 1 % for 1 min.	Hyoscine hydrobrom. 5 % for 1 min.	Atropine sulphate. 1 % for 1 min.
13 "	130	85	90	100
14 "	125	80	90	100
15 "	120	80	90	100
20 "	65	70	82	95
25 "	35	63	76	95
30 "	20	56	70	95

TIME.	(No. 665).	(No. 666).	(No. 355).	(No. 3,129).
0 min.	123	100	110	100
5 "	112	100	105	100
10 "	113	100	100	100
	Aconitine hydrochloride 0.33 % for 1 min.	Aconine hydrochloride 1 5/10 % for 1 min.	Physostigmine sulphate 2 % for 1 min.	Atropine sulphate 10 % for 1 min.
13 "	260	95	105	—
14 "	275	105	100	65
15 "	270	105	89	60
20 "	160	100	50	45
25 "	47	95	22	35
30 "	0	90	5	30

TIME.	(No. 840).	(No. 567).	(No. 724).	(No. 308).
0 min.	100	100	94	85
5 "	100	100	97	92
10 "	100	100	100	100
	Cocaine hydrochlor. 1 % for 1 min.	Morphine Tartrate 5 % for 1 min.	Ext. Opii. 5 % for 1 min.	Gelseminine 2 % for 1 min.
13 "	30	—	—	—
14 "	30	—	89	—
15 "	25	90	89	45
20 "	21	90	70	37
25 "	18	85	50	30
30 "	15	80	36	20

TIME.	(No. 3,122).	(No. 3,200).
0 min.	96	96
5 "	98	100
10 "	100	100
	Veratrine hydrochlor. 1 % for 1 min.	Veratrine hydrochlor. 0.1 % for 1 min.
13 "	62	114
14 "	20	116
15 "	0	120
20 "	0	114
25 "	0	86
30 "	0	50
35 "	0	30

THREE CASES OF NEW GROWTH WITH CAVITY FORMATION IN THE SPINAL CORD.

BY WILLIAM ALDREN TURNER, M.D., F.R.C.P.

AND

ASHLEY W. MACKINTOSH, M.D.

WE have been enabled, by the kindness of Dr. Ferrier, to record three cases of tumour of the spinal cord, which at one time or another during the course of the disease had been under his direct observation, either in King's College Hospital or at the Queen Square Hospital, and Cases 1 and 2 were exhibited and commented upon by him before the Neurological Society. The pathological examination of these cases has been carried out in the neuro-pathological laboratory at King's College, and we are indebted to Dr. Ferrier for the opportunities afforded us for this investigation.

CASE I.—Almost complete paralysis, with wasting of arms. Paresis of legs. Profuse general perspiration. Prominence of lower cervical and upper dorsal spines. Dissociation of sense of temperature from senses of touch and pain, practically limited to pectoral girdle. Death within two years of onset. New growth and cavity formation in cord, implicating both posterior columns and both posterior horns.

F. K., aged 18, a groom, was admitted to King's College Hospital under Dr. Ferrier, on April 5, 1890. The following is an abstract of notes taken at that time and during the subsequent course of his illness:—

There was nothing worthy of note in his family history, or in the patient's previous history.

Patient was quite well till about the end of February, 1890,

when his shoulders and upper arms began gradually to get weak, the right arm more quickly than the left. At the end of three weeks he could scarcely raise his arms at all. There was no history of any blow or fall, or exposure to cold and wet.

Condition on admission.—He is a healthy-looking, well-nourished lad. There is great weakness in abduction, flexion and extension at both shoulders; while adduction is well performed. The shoulders droop downwards and forwards, and are very feebly shrugged. The action of the pectorals and latissimi dorsi is feeble. Flexion and extension at the elbows are weak, but at the wrists fairly strong. Flexion of the fingers is weak and the grip feeble. The movements of the legs show normal power. In walking, his head droops forwards, and his gait is shuffling. Movements of the face are normal. The electrical reactions of all muscles are normal. The knee-jerks are increased. There is no ankle clonus. Patient can feel and localise correctly. The hands are congested and cold, and the face sweats a great deal. The cervical spines are abnormally prominent, but there is no pain on pressure over them.

July 14.—The loss of power in the arms has much increased. There is now complete loss of movement at both shoulders and elbows, while flexion and extension of the wrists and fingers are weak, especially on the right side. He cannot abduct the second and third fingers. There is great wasting of all the scapular muscles, including the serratus magnus on both sides. The hands are cold and flushed. The face is flushed and still sweats. Occasionally there is a feeling of "pins and needles" in the feet, and pains in both arms. The legs have been feeling rather stiff for a few days. Once, while walking, patient felt a pain in the right leg along the external saphena vein, and the leg gave way, and he fell. From this time onwards, the legs have become weaker, especially the right.

October 2.—The hands are cold and livid. There is no power of movement in either arm, except weak supination of the right forearm, and feeble extension of the fingers of both hands. All the paralysed muscles react to faradism, the pectorals and triceps of both sides rather feebly. There is no reaction of degeneration. In all muscles K.C.C. is greater than A.C.C.. The left pupil is dilated.

January 30, 1891.—Patient was admitted to Queen Square Hospital under Dr. Ferrier. He states that his gait has gradually become worse, till now he cannot walk without assistance. For some time he has had gradually increasing difficulty in swallowing,

and occasionally some difficulty in passing water, but never incontinence.

Present condition.—There is some weakness of the abdominal muscles. He cannot raise himself up in bed, or sit up when raised. In deep inspiration the upper part of the thorax is elevated, but there is very little expansion. There is a small amount of movement at both shoulders and elbows, but practically none at either wrist-joint. There is some power of flexion and extension of the fingers, especially of the left hand, and chiefly of the thumb. The left arm is rather rigid. The movements of the legs are fairly good when in bed, but there is weak dorsal flexion of the ankles when standing up. Both legs are somewhat rigid, especially the left. The gait is feeble. He cannot walk without assistance, and drags both legs very much. He sways when standing with feet close together, and especially when his eyes are shut.

All muscles of arms and legs react normally to faradism, except that the re-action of the supra-spinati and infra-spinati is feeble. All muscles react normally and readily to galvanism, but the forearm-muscles show less than the normal difference between K.C.C. and A.C.C.

Sensation of all forms is normal, except that thermic sensibility is deficient on both arms from the elbows downwards. Sense of position of limbs is normal. The knee-jerks are equal and much exaggerated; there is no ankle clonus. No wrist or elbow-jerk can be obtained on the right side, but there is a distinct elbow-jerk on the left side. No plantar reflex can be elicited.

Trophic Changes.—There is marked wasting of both deltoids, and of the infra-spinati and supra-spinati; but no distinct wasting of the pectorals, trapezii or latissimi dorsi. The legs are not wasted. The face is flushed. Patient always feels hot, and his whole body is constantly bathed in sweat. His hands very quickly get blue and cold when exposed. There is a marked projection backwards of the spinous processes from the seventh cervical to the fourth dorsal spine, but no pain on pressure or on movement. There is slight nystagmus on lateral deviation, especially to the right. All ocular and pupillary movements are normal. The pupils are dilated, and the left is rather larger than the right. Swallowing, especially of solids, is somewhat difficult.

July 10.—The left pupil is much larger than the right. There is considerably more movement in both arms, especially the right.

Sensation.—Tactile sensibility is normal everywhere; but there is decided impairment of thermic sensibility on the neck and upper part of the trunk, extending down almost to the level of the umbilicus, and on both arms, especially the right.

October 23.—*Sensation.*—Tactile and algesic sensibility is still intact, but there is no appreciation of either heat or cold on the neck and trunk down to rather above the level of the umbilicus. The same is true of the whole of the right arm, and of the left arm down to the elbow, while there is imperfect appreciation of temperature on the left arm below the elbow. Below the area of thermo-anæsthesia, on the trunk, there is a zone where cold is appreciated, but not heat. All the muscles of the arms react to a moderate faradic current, but the deltoids require a somewhat stronger current than the other muscles. All the muscles react normally and readily to the galvanic current. The arms are rather weaker again. The left pupil is still larger than the right.

December 3.—There is still profuse general perspiration; the face is flushed and markedly injected, the hands are livid and cold to the touch. The wasting of the shoulder-muscles is now very great, especially on the left side. There is marked wasting of the interossei and of the thenar and hypothenar eminences. There is still some power of flexion and extension of the fingers, but only very slight movement at the wrists and elbows. No movement is possible at the left shoulder, except what is due to the pectoralis major, while there is a fair power of adduction, but no abduction at the right shoulder. There is still a considerable amount of power in the legs.

Both knee-jerks and plantar reflexes are enormously exaggerated, especially the right, and there is well-marked ankle clonus on both sides. There is incontinence of urine and fæces, but patient is conscious of the necessity. There is obstinate constipation. Nystagmus is now distinct in both eyes on lateral deviation. The pupils are now equal and dilated, but react normally. The function of all other cranial nerves is normal.

Tactile sensibility is normal everywhere, except that it seems rather defective towards the lower edge of the ramus of the jaw; and there is on the anterior aspect of the trunk a well-defined semi-lunar area of anæsthesia, the upper margin of which passes from nipple to nipple, about one inch below the xiphoid, and the lower margin through the umbilicus. The legs are hyperæsthetic.

Algesic sensibility is everywhere normal.

Thermic Sensibility.—From the chin anteriorly, and a little below the line of hair posteriorly, down to the level of the umbilicus both anteriorly and posteriorly there is complete thermo-anæsthesia; except that on the area above the second rib in front, extending up between the sterno-mastoids, all thermic sensations are called cold, and there is a slight appreciation of heat and cold

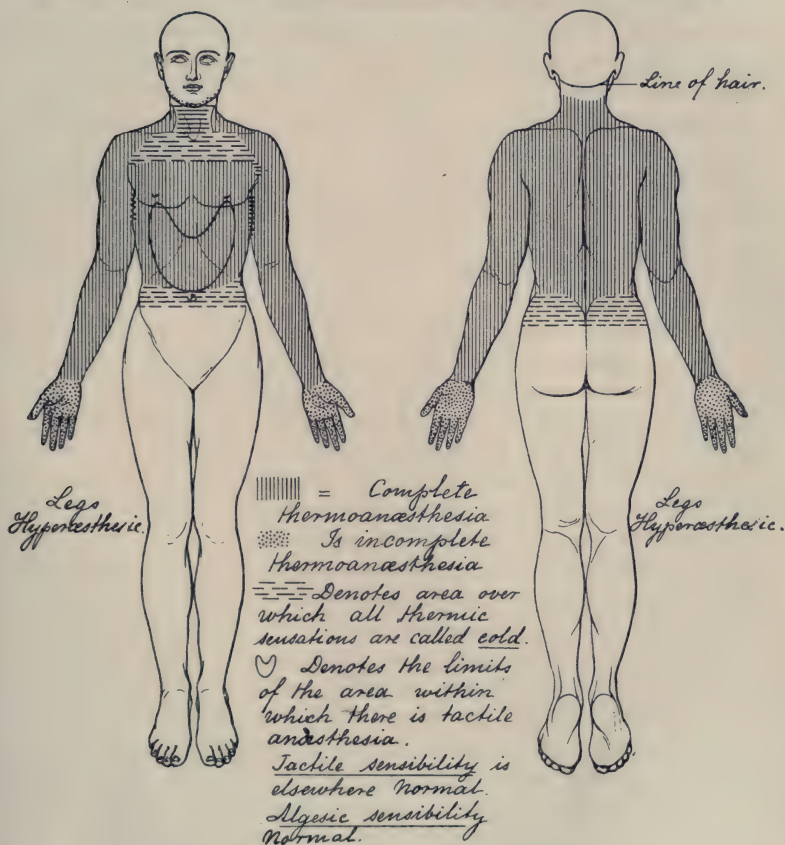


CHART 1.

in both axillæ. Below the area of thermo-anæsthesia on the trunk, there is a zone about two inches in depth on which all thermic sensations are called cold. Below this, the trunk and legs have normal thermic sensibility. On the arms there is complete thermo-anæsthesia down to the wrists, while there is diminished appreciation of temperature on the hands

Muscular sense is normal in all the limbs.

January 14, 1892.—Patient began to suffer from difficulty in breathing a week ago. His temperature rose, and he became much weaker, and died to-day.

There was influenza in the wards.

At the *autopsy* it was found that there was no angular curvature of the spine. The cord in the cervical region was enormously enlarged, and filled practically the entire vertebral canal—the medulla appeared to be implicated in the enlargement. In the upper part of the cervical region the cord was found, on section, to consist almost entirely of a dark pulpy mass, surrounded by a mere rim of nerve tissue. The amount of central new growth gradually diminished lower down, and, at the various levels of the cord, presented the same relations and the same extent of cavity formation as will be found in the detailed description of the microscopical appearance of the sections of the cord.

MICROSCOPICAL EXAMINATION OF THE SPINAL CORD.

Mid-cervical Region (fig. 1).—At this level the cord measures 21 m.m. transversely, by 16 m.m. antero-posteriorly. Each posterior column in its anterior two-thirds is occupied by a new growth, composed of laminae concentrically arranged; the structures of the cord in the neighbourhood are much compressed, so as to cause an alteration both in shape and position. The grey commissure is retained. The lumen of the central canal is still present; its ventral side is lined by several layers of endothelium, which, from the form of the cells and their method of staining, look like normal lining cells; but the dorsal side of the canal is entirely bereft of the normal lining, and the lumen is broken into by a large number of round and spindle-shaped cells. In some of the sections from this region there is, dorsally and laterally to the central canal, a small area containing an enormous increase in the number of cells, amounting actually to a third tumour. The intermediate grey matter is much broken up, especially on the left side, due, apparently, to infiltration of gliomatous tissue. The anterior horns are normal and unaffected by the tumour-growth, the large ganglion cells presenting normal features. The left posterior horn is compressed, but not directly implicated by the new growth; the right posterior horn is distinctly involved in the growth, and in it there lies a cavity surrounded by gliomatous tissue.

Each tumour is composed of a slightly fibrillated neuroglial tissue, with few cells in its central part, but distinctly more

cellular towards the periphery; the cells are round or spindle-shaped. In the centre of the left tumour mass a recent hæmorrhage has taken place, while in the right tumour there are several small cavities, each containing a homogeneous fibrinous material, evidently the remains of a hæmorrhagic clot. These cavities, as well as that in the right posterior horn, are smooth-walled, but there is no trace of an endothelial lining. In the peripheral parts of each tumour, and in the adjacent nerve tissue, there is a great increase in the number and size of the capillary blood-vessels and of the smaller arterioles.

There is very marked degeneration in the whole of the antero-lateral region of the cord on the left side; while on the right side there is slight degeneration in the crossed pyramidal tract, extending forward in the antero-lateral periphery of the cord to the region of the direct pyramidal tract, where the degeneration is much more marked.

Lower Cervical and Upper Dorsal Regions (figs. 2 and 3).—The appearances in these two regions blend with one another, and therefore a continuous description is given.

(1) In the first of the sections at this level (fig. 2), the tumour-mass in the left posterior column is again seen, but reduced in size, and retaining its circular appearance. The definite tumour-like appearance on the right side has disappeared, and is replaced by an elongated oval tract of nerve tissue, apparently occupying the anterior two-thirds of the zone between the posterior horn and the posterior column, extending into the grey commissure, and containing a slit-like cavity partially filled with a fibrinous material. In the posterior half of the left posterior horn there is another slit-like cavity, surrounded by a thin layer of new tissue; while anterior to this, and in the most ventral and external part of the posterior column, there is another slit-like cavity also filled with fibrinous material, its anterior extremity passing into the grey commissure. The walls of all these cavities are very sharply cut, but in none is there a trace of an endothelial lining. The intermediate grey matter is broken up, especially on the left side, and in the spaces the same fibrinous material is seen. The central canal is represented by a mass of small round cells.

(2) In a section a little below the last, exactly the same appearance is seen, except that the tumour-mass in the left posterior column presents a clear-cut cavity in its centre.

(3) In this section, a little lower still, four cavities are now seen (fig. 3)—one in the posterior part of each posterior horn, and

one in the anterior part of each posterior column. Each of the cavities is very clearly cut, and is surrounded by neuroglial tissue, very small in amount, except around the cavity in the left posterior column, which is enclosed in a thick, dense mass of neuroglial tissue. In this region, the intermediate grey matter seems to be undergoing myxomatous degeneration.

There is degeneration in both crossed pyramidal tracts, in the antero-lateral periphery of the cord, especially of the right side, and in both direct pyramidal tracts—degeneration is specially distinct in the last.

Mid-Dorsal Region.—At this level, the cavities are limited to the posterior horns, one on each side. They present the character of clean-cut cavities, surrounded by a dense layer of neuroglial tissue without a trace of an endothelial lining; by their anterior extremities they extend well into the intermediate grey matter, and posteriorly they reach almost to the tip of the gelatinous substance of Rolando. The cavity on the left side contains homogeneous fibrinous material.

Lower Dorsal and Lumbar Regions (figs. 4 and 5).—In all sections made in these regions, the same features are observed—viz., a single cavity, surrounded by a layer of neuroglial tissue, in the anterior two-thirds of the left posterior column, close to the posterior horn, but distinctly separated from it by nerve fibres. The only difference at the several levels is the shape of the cavity. At the level of the eighth dorsal nerve roots, the cavity is an antero-posterior slit; in the lower dorsal region (fig. 4) it has a spur-like process extending outwards; while in the lumbar region a similar process passes inwards as well, so that the cavity presents a crucial appearance. In the lower lumbar region (fig. 5) there is the same appearance, with the addition of a small localised gliosis, occupying quite the anterior part of the left posterior column; this is separated from the cavity by a well-marked strand of nerve fibres. In one of the sections this gliosis shows clear signs of cavity formation. In all these sections the central canal is represented by a mass of small cells, and the posterior commissure is normal. In the mid-dorsal and lower-dorsal regions there is well-marked degeneration in the pyramidal tracts of both sides, direct and crossed; in the lumbar region the degeneration is limited to the crossed pyramidal tracts.

We are indebted to Dr. James Taylor for the description of the macroscopical appearance of the cord, and for the sections from which the microscopical examination was made.

REMARKS.

In this case, points of clinical interest are the comparatively rapid course—death taking place, within two years of the onset of symptoms—and the dissociation of the sense of temperature from the senses of pain and touch.

The new growth and cavity formation, as figs. 1-5 show, involve the grey commissure, the intermediate grey matter, the posterior horns and the anterior two-thirds or so of the posterior columns, especially in their external parts. Fig. 1 shows two distinct tumour-like masses in the posterior columns, that on the right side implicating the posterior horn as well. From the concentric arrangement of the laminæ in the new growths, and from the fact that the grey commissure is present, it appears likely that the growths originated in the posterior columns, especially in their external parts, or in the adjacent posterior horn.

There is a direct transition from the true tumour-like formation in the mid-cervical region (fig. 1) to the appearance presented in figs. 2 and 3. The tumour-mass on the right side in fig. 1 is converted into the mass of gliosis, seen on the right side in fig. 2 occupying the same region, viz., the external part of the posterior column and the adjacent part of the posterior horn, while, in a section made a little lower (fig. 3), this same region shows two smaller masses of gliosis, each with a clear-cut cavity in its centre, the posterior lying in the middle of the posterior horn and the anterior occupying the region between the anterior part of the posterior horn and the posterior column. Lower still, about the mid-dorsal region, the gliotic process on the right side is strictly limited to the posterior horn, and gradually ends there, without showing at any part a direct communication with the region of the central canal.

Similarly, the growth seen on the left side in fig. 1 loses its distinct tumour-like appearance, but more gradually than on the right side: thus in fig. 2 it is still quite like a tumour, and there is now a distinct cavity in its centre, continuous with the cavity formed by the hæmorrhage in fig. 1. But there are also two pieces of gliosis on the left side in fig. 2,

each with a clear-cut cavity, both in the posterior horn or adjacent part of the posterior column, the anterior reaching well into the grey commissure, but not communicating with the region of the central canal. In fig. 3 the growth in the left posterior column has now lost the tumour-like appearance, and consists of a thick mass of gliosis, with a very clearly-cut cavity in its centre; there is now only one cavity in the left posterior horn, corresponding in situation with the posterior of the two cavities in fig. 2. Still lower, in the mid-dorsal region, the gliosis and cavity formation on the left side become limited to the posterior horn; while in the lower dorsal and lumbar regions the morbid process moves gradually towards the adjacent part of the posterior column, leaving the posterior horn intact (figs. 4 and 5). There is absolutely no trace of an endothelial lining to any of the cavities, despite their remarkably clear-cut appearance. At no part is there a dilated central canal; it is represented in almost the whole dorsal and lumbar regions by a mass of nuclei.

The gradual and direct transition between the true, tumour-like appearance, or *gliomatosis*, in the sense of Hoffmann,¹ seen in fig. 1, and the appearance presented in fig. 3, is very striking; the latter presents all the signs of a gliosis or proliferation of the neuroglia. There can be no doubt that the corresponding cavities are continuous throughout.

The fact that a fibrinous material is found in many of the cavities, points to a hæmorrhage as their cause, and there is an undoubted hæmorrhage on the left side in fig. 1. At other parts there is an appearance of myxomatous degeneration, leading to cavity formation.

Attention may be drawn to the fact that the morbid process, when traced to its lower limit, ends, not in the region of the central canal, but either in the posterior horn (as on the right side, *cf.* also Case 2), or in the adjacent part of the posterior column (as on the left side); so that if any deduction can be drawn from the mode of termination as to the starting point of the process it

¹ Hoffman. *Deutsche Zeitschr. f. Nervenheilk.*, 1893, iii., s. 1-136.

would be against the view of a central origin (*i.e.*, an origin around the central canal), and in favour of the view already drawn from the appearance presented by fig. 1, *viz.*, that the growths started in the posterior column, or adjacent parts of the posterior horns.

We have been unable to trace any connection between the small mass of gliosis in the anterior part of the left posterior column in the lower lumbar region (fig. 5), and the larger gliosis behind it, or the gray commissure in front of it, so that it seems quite an isolated piece of gliosis.

The symmetrical affection of the two sides of the cord, and the bilateral descending degeneration, are in accordance with the fact that the symptoms were bilateral and symmetrical; this presents a contrast to Case 3, in which it will be seen that the lesion and the symptoms were mainly unilateral.

CASE II.—*Almost complete paralysis with wasting of arms. Paresis and rigidity of legs. Hyperidrosis of face and arms. Marked pain and tenderness in upper part of spine. Loss of sensation over pectoral girdle, at first thermo-anæsthesia only, to which was added later analgesia, and finally anæsthesia: legs slightly affected before death. Death within one year of onset. New growth (neuroglioma ganglionare), and cavity formation in upper part of cord, involving mainly posterior columns and posterior horns.*

A. P., aged 23, a 'bus-driver, was admitted into King's College Hospital, under Dr. Ferrier, on June 5, 1894. The following is an abstract of notes taken at that time and subsequently:—

Family History.—Patient's father and two brothers died of heart disease, and an aunt had a stroke of paralysis. No tubercular history.

Previous History of Patient.—Patient has been much exposed to cold and wet. There is an indefinite history of an injury to the spine ten years ago. Three years ago patient had "influenza," and suffered from occipital pains for six months afterwards. No specific history.

History of Present Illness.—About the end of February, 1894, on getting off his 'bus one night, patient was seized with severe

pain between the shoulders. His neck felt stiff, and he had difficulty in straightening his back and in using his arms. This condition passed away in about an hour, but ever since that time, patient has had attacks of pain shooting from his neck down both arms and up to his occiput, and he has complained of stiffness of the neck, and of a feeling of "pins and needles," and numbness in the region of the pains.

Condition on Admission.—Patient is a well-nourished, healthy-looking man. He holds his head and neck very stiffly, and any forcible attempt to move them causes pain. Under an anæsthetic there is found to be some resistance to movement of the head to the right. All movements of the arms are present, but weaker than normal. He can stand quite steadily with eyes shut. The knee-jerks are present and equal. There is no ankle-clonus.

SENSATION.—*Subjectively*, there is a burning pain round the right side of the upper part of the chest, and a sharp pain shooting down the right arm. *Objectively*, tactile and algesic sensibility is normal. There is tenderness on pressure over the upper part of the spine extending down to the second dorsal spinous process. The function of all the cranial nerves is normal.

After the above date, there was a temporary improvement in all the symptoms, especially the pains, but later the pains in his neck and arms and between the shoulders returned with increasing severity, and both arms began to get weak, the left one first. On August 15, 1894, patient entered University College Hospital, under Dr. Bastian. The following is an abstract of notes of his condition, made by Mr. C. Atkinson, and kindly supplied to us by the Registrar:—Movement of the head is limited in all directions owing to the pain caused thereby in the region of the cervical spine. Flexion of the trunk is limited. There is marked weakness of both arms, especially the left. At the shoulders the power of abduction is lost on both sides, flexion is lost on the left side, and limited to 30° on the right side, while rotation also is lost on the left side. At the elbows, there is some power of flexion on the right side, none on the left. Movement at the wrists and fingers is normal. The legs retain power of strong movement at all joints. The right knee-jerk is very brisk, but there is no ankle-clonus. *Sensation* of all forms is normal except that thermic sensibility is somewhat erratic on both hands, especially on the forefingers, and there is defective appreciation of heat over the upper part of the trunk; subjectively there is pain in the region of the upper dorsal spine, radiating down the right arm and up towards the occiput. There is marked hyperidrosis of face, hands and feet. The right pupil is larger than the left.

Subsequent Course.—The arms and legs became progressively weaker, the legs got rigid, the knee-jerks much exaggerated, and ankle-clonus well marked. Breathing became almost entirely thoracic, and occasionally the abdomen was retracted during inspiration. The pains continued very severe, but shifted their position lower down the dorsal spine. There was an almost constant feeling of extreme cold over the left shoulder and left arm down to the elbow, and over the left side of the trunk, and there was a marked girdle sensation round the chest below the level of the nipples. The spine remained tender on pressure, and the area of tenderness gradually extended down as low as the mid-dorsal region. The hyperidrosis of face and hands remained very constant. The muscles of the arms, forearms and hands gradually wasted to an extreme degree, but all retained a fairly normal reaction to the faradic current, although it was noted that A.C.C. was greater than K.C.C. as early as October 1. On October 29, a small circular ulcer, of the size of a split pea was noted over the head of the metatarsal bone of the right thumb, and a few days later a similar ulcer was found on the left thumb. Constipation remained obstinate throughout.

THE AFFECTION OF SENSATION gradually increased. Thus on September 9 it was noted that tactile and algæsic sensibility was normal everywhere, while on the right hand up to the wrist, on the left hand up to the elbow, and on the chest from the third rib down to the costal margin *all* thermic sensations were described as cold.

The condition as regards sensation, was noted as follows, on October 11.

Tactile sensibility appears normal, except that patient cannot readily discriminate different forms of tactile impression.

Algæsic sensibility is considerably blunted, but not lost, over the front of the chest, from the second intercostal space to the costal margin and over both arms, except over the outer part of both upper arms.

Thermic sensibility is defective over the whole of both arms, and over the trunk, from the level of the second intercostal space to the umbilicus.

The condition of sensation on October 31, was the same as in the last note, except that the area of defective algæsic and thermic sensibility now extended on the trunk, down as far as Poupart's ligament, and the right arm had now quite lost the sense of pain, while thermic sensibility was quite gone on both arms.

On November 24, when patient left University College Hospital,

it was noted that tactile sensibility seemed distinctly deficient over both arms, and over the anterior part of the chest, while there was an extension of the complete loss of thermic and algesic sensibility to the upper part of the trunk, and there seemed to be a slight defect in appreciation of difference of temperature in the legs.

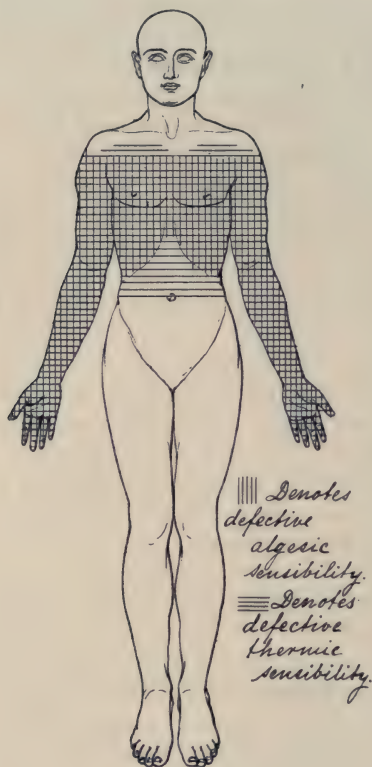


CHART 2.

The general condition of patient on re-admission into King's College Hospital on November 26, was as follows :—There is still marked hyperidrosis of face and arms, and subjective sensation of cold over the arms and abdomen. There is practically no abdominal respiration, and breathing is often very difficult. There is no power of movement in either arm, except some elevation of the shoulders, and some flexion and extension of the right wrist, and of the fingers of both hands. The legs are drawn up and rigid ;

flexion at the knees is very weak, and movement of the ankles and toes is quite lost, except some slight movement of the halluces. All the muscles of the arms are much wasted; but all react to faradism, although the reaction of the deltoid, pectoralis major, triceps, and supinator longus of both sides is feeble. The knee-jerks are much exaggerated, especially the right; ankle clonus is well marked. There is constant pain down the whole length of the spine, but especially over the cervical and upper four or five dorsal spines; the pain is increased on movement, especially on sitting up in bed. There appears to be a slight irregularity of the upper two cervical spines. Movement of the head to the right is very limited on account of the pain. *Sensation* is as noted on November 24; the sense of position of the arms and fingers is not clear. The right pupil is rather larger than the left, and the tongue deviates slightly to the left. The function of other cranial nerves is normal.

December 13.—Sensation of touch is distinctly defective on the trunk, above the umbilicus.

Patient's condition remained unchanged till January 27, 1895, when his breathing suddenly became laboured; loud râles were heard in the chest, breathing got worse and pulse weaker, and he died on January 28.

At the *autopsy* it was found that the spinal cord was enormously enlarged and abnormally soft in the cervical region, filling practically the entire vertebral canal. The medulla did not appear to be implicated. The vessels of the dura mater of the cord were greatly distended. On section, the upper and middle cervical regions appeared to be quite filled with a soft, pulpy, reddish-brown mass, which bulged out at the cut ends. This abnormal appearance gradually diminished lower down, becoming more and more confined to the central region and posterior columns, till about the mid-dorsal region, where the cord appeared normal.

Microscopical Examination of the Spinal Cord.—The MEDULLA appears perfectly normal, except that it is abnormally vascular, especially in its posterior half, and at its lower part there is a distinct small-cell infiltration underneath the ependyma of the fourth ventricle.

At the level of the second and third cervical nerves (fig. 6) the central part of the cord is seen to be occupied by a new growth, which is circumscribed with remarkable clearness. It occupies the anterior three-fourths, or more, of the posterior columns and the grey commissure. There is no trace of a central canal, nor can the anterior commissure be seen, *i.e.*, the tumour growth

comes right up to the antero-median fissure. The intermediate grey matter, and both posterior horns, are very much compressed, and are directly invaded by the new growth, which extends along the posterior horns to the periphery of the cord. The grey matter of the anterior horns is also very much compressed, and its cells are smaller and fewer than normal. Degeneration is fairly well marked in the periphery of both sides of the cord, in the region of the direct cerebellar tract, and of the antero-lateral tract of Gowers, but there is very little sign of degeneration in what remains of the posterior columns. The new growth consists of small neuroglia-like cells, round or rather spindle-shaped, with deeply stained nuclei, and numerous fine processes; the cells are much more densely packed at the periphery than in the central part of the growth. A striking feature is the great amount of vascular development in the new growth, especially at its periphery, and in the nerve-tissue immediately outside it. The vessels are very numerous; and many have thick, homogenous walls which stain very poorly. The central parts of the tumour have undergone a metamorphosis, partly fatty, as shown by osmic acid, partly myxomatous, as shown by spaces containing clear, homogenous tissue.

At the level of the fifth cervical nerves (fig. 7), the new growth reaches its maximum in point of size, filling the whole cord with the exception of a marginal rim which is nowhere thicker than $1\frac{1}{2}$ mm. The structure of the new growth is the same as at the last level, and its relations are also unchanged, allowance being made for its increase in size. The anterior horns are now very much compressed, but the ganglion cells are still quite visible, and have fairly well-marked processes. The anterior fissure is greatly displaced, and very little can be seen of the posterior horns. There is no distinct tract of degeneration in the rim of nerve tissue.

At the level of the sixth and seventh cervical nerves (fig. 8), the abnormal tissue has now considerably diminished in amount, and is confined to the posterior commissure, the intermediate grey matter, and the anterior two-thirds of the posterior columns. Posteriorly there is no sharp line of demarcation between the new growth and the normal nerve tissue. The anterior commissure now appears normal, and the central canal is distinct, but its wall is rather broken up. The anterior horns appear normal.

There are distinct cavities in the new growth, two in the anterior half of each posterior column. Those on the right side have a sharp, sinuous margin, being lined by a layer of wavy

tissue containing many deeply-stained nuclei; they are filled with a fibrin-like material. The cavities on the left side present a contrast to those on the right side, in that they have not such sharply-cut margins, their walls present a broken-down appearance, and they do not contain fibrin-like material. There is no trace of an endothelial lining in any of the cavities. The new growth consists mainly of small cells similar to those seen at higher levels, but very conspicuous now are larger bodies seen throughout the whole of the new formation at this level, but most numerous in the region of the intermediate grey substance. They vary considerably in size and shape (fig. 9). The largest are about the size of the ganglion cells of the anterior horn. Many are round or oval, others are rather irregular in shape. Their nuclei are large and stain deeply, in some there are two or more nuclei. These bodies are surrounded by a narrow clear space, and no trace of fine fibrillary processes can be detected arising from them. They stain in quite the same manner as ganglion cells.

There is no distinct tract of degeneration at this level, but there is, perhaps, a diffuse hyperplasia of the supporting tissue of the whole cord.

At the level of the eighth cervical nerves, the amount of new growth has still further diminished, the cavities in the right posterior column have disappeared, and there is now only a very slight amount of new tissue in the anterior part of the right posterior column, quite cut off from the region of the central canal. There is still a cavity in the anterior third of the left posterior column, surrounded by abnormal tissue which is connected with the posterior commissure. Both posterior horns are normal.

At the level of the first dorsal nerves the condition is the same, except that the gliotic tissue surrounding the cavity in the anterior part of the left posterior column is now quite cut off from the grey matter of the cord by normal nerve fibres, so that the posterior commissure and central canal region are quite normal.

Lower down still, the cavity in the left posterior column elongates antero-posteriorly, and tends to go more and more outwards till it invades the left posterior horn as well as the adjoining part of the posterior column, and the right posterior horn, which was quite normal at higher levels, presents a distinct nuclear infiltration which increases as we descend and develops a cavity in its interior.

At the level of the third dorsal nerves, there is a cavity on

either side strictly confined to the posterior horns, and not connected with the grey commissure; they are surrounded by a very small amount of nuclear tissue. These cavities gradually diminish lower down, till about the level of the fourth or fifth dorsal nerves, both cavities and abnormal tissue have disappeared, and the posterior horns and posterior columns become normal. In the upper and middle dorsal regions, but not in the cervical or lower dorsal region, the central part of the grey matter, and, to a less extent; the adjacent white matter show by Marchi's method of staining a vast number of black masses and dots (fig. 10), each larger mass being a conglomeration of smaller particles. Carmine-stained sections at the same levels show homogeneous colloid-like masses varying in size, and corresponding very closely in size and situation to the black masses seen by Marchi's method. There is no appearance of a nucleus in them. At these levels also, sections stained by the Weigert-Pal method, show that the grey matter is markedly deficient in the normal fibrillary nerve plexus.

From the level of the mid-dorsal region downwards, there is no abnormal tissue or cavity formation anywhere, and there is no sign of descending degeneration. The cord, in fact, seems perfectly normal. We shall make some remarks later as to the condition of the central canal.

REMARKS.

The rapidity of the course—death within one year of the onset of symptoms—the marked tenderness of the cervical spine, the severity and persistence of the pains in the back of the neck, and shooting down the arms, and the progressive affection of the different forms of sensation—temperature, pain, touch, in the order named—are important clinical points in this case.

The enormous size which the new growth has attained at its maximum in the mid-cervical region, as shown in figs. 6 and 7, and the great amount of compression and distortion of the cord are very striking, especially when taken in conjunction with the fact that there is no distinct trace of secondary descending degeneration in the lower parts of the cord.

The part of the cord occupied by the new growth is the same as in Case 1 (*i.e.* the central region, the posterior

horns and the front of the posterior columns), but in this case the growth extends forward to the antero-median fissure (figs. 6 and 7) so that there is no trace of an anterior commissure.

On being traced downwards, the morbid process gradually assumes the appearance of a gliosis rather than that of a tumour, and becomes limited to the anterior parts of the posterior columns, while the region of the central canal becomes normal. The gliosis on the left side tends to move more and more outwards, so as to implicate the posterior horn, and finally it is restricted to the posterior horn where it ends about the level of the fourth or fifth dorsal nerves. On the right side the gliosis in the posterior column gradually ends at the level of the first dorsal nerves, but here the right posterior horn, which appeared quite normal at higher levels, begins to show a small cell infiltration, which gradually increases as we descend, develops a cavity in its centre, and ends in the posterior horn at the level of the fourth dorsal nerves: this piece of gliosis in the right posterior horn seems to be quite isolated.

This case therefore resembles Case 1 in showing, first, that there is a direct transition from a distinct tumour-like mass to a process of gliosis, and secondly that the morbid process terminates below, not around the central canal, but in the region of the posterior columns.

None of the cavities show any trace of an endothelial lining, and they nowhere communicate with the central canal. The new growth appears to have undergone various forms of degeneration, all of which no doubt tend to cavity formation. At some parts there is a fatty metamorphosis, as shown by osmic acid; at other parts there is a myxomatous change, and this is specially seen in the walls of the vessels, many of which are very thick, and appear remarkably homogeneous. The enormous development of vessels in and around the new growth is one of the most striking features: the abnormal state of the vessels—the thick, homogeneous walls and the narrow or obliterated lumen—especially towards the centre of the growth, must lead readily to cavity formation, by softening from malnutrition and absorption of the *débris*. The

cavities seen in fig. 8 have almost certainly an origin of this nature; and the difference between the irregular, broken-down walls of the cavities on the left side, and the comparatively sharp cut margins of those on the right side is probably a difference in the stage of absorption of the broken-down *débris*. The presence of a fibrin-like material in some of the cavities, suggests that hæmorrhage assists in the formation of these spaces. The condition of the central canal varies: at the levels from which figs. 6 and 7 were taken there is no trace of it, while at the level of fig. 8 it presents an appearance as typically normal as we have ever seen; *i.e.*, it is lined by a single, uninterrupted layer of cylindrical epithelial cells, it is not larger than usual, and there are only a few nuclei around it. On the other hand, many sections taken from the dorsal or lumbar region show a central canal larger than usual, but still probably within the normal limit, with the single layer of cylindrical lining cells interrupted at some parts, and a slight proliferation of small cells around it. Comparison with the central canals of a large series of cords, normal and morbid, in which there was no suspicion of such a condition as hydromyelia or syringomyelia, has led us to the conclusion that there is no part of the central canal of Case 2 which can be called undoubtedly abnormal: it is very difficult, however, to lay down the limits of a normal central canal.

With regard to the nature of the new growth, we believe it is the result of the proliferation, not only of the neuroglia, but also of the true nervous elements. The large bodies (fig. 9), which were described as occurring in the new growth, about the level of the sixth and seventh cervical nerves, must, we think, be of the nature of ganglion cells, and not neuroglia cells, for the following reasons:—some are as large as the ganglion cells of the anterior horns, and have large nuclei; there is an entire absence of the fibrillary network of processes so characteristic of neuroglia cells; they occur in greatest numbers in the region of the intermediate grey substance; and their method of staining is identical with that of ganglion cells. They do not, however, show the nucleolus usually so distinct in ganglion cells, nor do they

contain pigment granules. There are also a few nerve-fibres, rather varicose or moniliform in appearance, scattered throughout the growth at this level. We believe, then, that this is an example of that rare form of tumour, known as *neuroglioma ganglionare* or *cellulare*, a name first suggested in connection with a case reported by Baumann.² The description and figure given by Ziegler³ correspond very closely to the condition in our case. In the case recorded by Baumann, there were two tumours of the cerebrum, of which the most striking features were large cells, possessing the characters of ganglion cells, and an abnormal number and arrangement of nerve-fibres: this is, however, rather a case of heterotopia of the grey matter than a true *neuroglioma ganglionare*. The only examples of the latter, which we can find recorded, are those of Klebs,⁴ who describes *neurogliomata* of the cerebrum, cord, and peripheral nerves; two cases of *neurogliomata* of the cord are recorded at pages 75 and 77, the most conspicuous features being large ganglion cell-like bodies, and broad bands possessing the character of hyperplastic nerve-fibres. Although we cannot agree with his conclusion as to gliomata in general, viz., that they "arise *mainly* from the true nervous elements of the central nervous system," we believe that a true *neuroglioma ganglionare* may occur, just as a true *neuroma* may.

We have seen that in the upper and mid-dorsal regions carmine-stained sections showed homogeneous bodies, and sections stained with osmic acid, showed black masses and particles, especially in the region of the central grey matter (fig. 10). These appear to be due to the same phenomenon, viz., a fatty degeneration of the nervous elements, and this is in accordance with the fact noted by the Weigert-Pal method of staining, that there is a great deficiency of the normal fibrillary nerve plexus in the grey matter of this region.

² Baumann. "Beiträge v. Zeigler," II., 1888, s. 500-508.

³ Ziegler. "Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie," 8. Auflage, Bd. I., s. 417-418.

⁴ Klebs. "Präger, Vierteljahrsschr. f. prakt. Heilkunde," 1877, s. 1.

CASE III.—*Left hemiparesis; right leg weak; no wasting of muscles; analgesia and thermo-anæsthesia of nose and ears; loss of all forms of sensation on right half of body (except face); patchy dissociation of touch from pain and temperature on left half of body—left arm and left leg below knee unaffected; sweating only on right side; small recurring growths on ring fingers; cervical spine concave to right. Death within four years of onset. New growth and cavity formation in cord implicating the ventral part of both posterior columns and the left posterior horn; marked degeneration of crossed pyramidal tract and antero-lateral descending tract of left side.*

T. H., aged 25, a coalporter, was admitted into King's College Hospital, under Dr. Ferrier, on July 24, 1892, complaining of difficulty in walking and weakness of the left arm. The following is an abstract of notes taken at that time and during the subsequent course of his illness:—

The family history shows nothing worthy of note.

Patient never suffered from any illness before the onset of the present symptoms. At the age of 5, he was knocked down by a cart, the wheel passing over the left shoulder and arm without causing a fracture.

History of present illness.—About July, 1889, when going to work one morning he suddenly felt his left arm “go to sleep,” the numbness commencing in the hand. As this numbness continued for several weeks he was admitted into the West London Hospital, and underwent an operation there on the supposition that “something had formed under the biceps”; this gave no relief. After this he resumed work, but about a year later he suddenly lost almost entire power of the left arm. Under faradic massage, the arm regained strength sufficient to enable him to resume work. About three months later, he lost power in the left leg, the paresis coming on rapidly. His right leg also became weak, and it was found at this time that he had lost almost entire sensibility to touch and pain on the whole right half of the body, except the face. On one occasion he tore open his right hand very badly without feeling it.

Condition on admission is noted as follows:—Patient is a healthy-looking, well-nourished man. He stands with his shoulders and head bent forwards, and stoops towards the right side owing to a slight lateral curvature of the spine with concavity towards the right, the left shoulder being distinctly higher than the right. The chin droops on the chest from weakness of the extensors of the neck. There is marked weakness of the

muscles of the left shoulder girdle, and paresis of both legs. He cannot dorsally flex the left ankle. He walks with left leg very stiff, and dragging his left foot on the ground. He cannot toe the line or walk steadily with eyes shut. All muscles react readily to faradism. The left trapezius and biceps react less strongly, but the left deltoid rather more strongly than the cor-

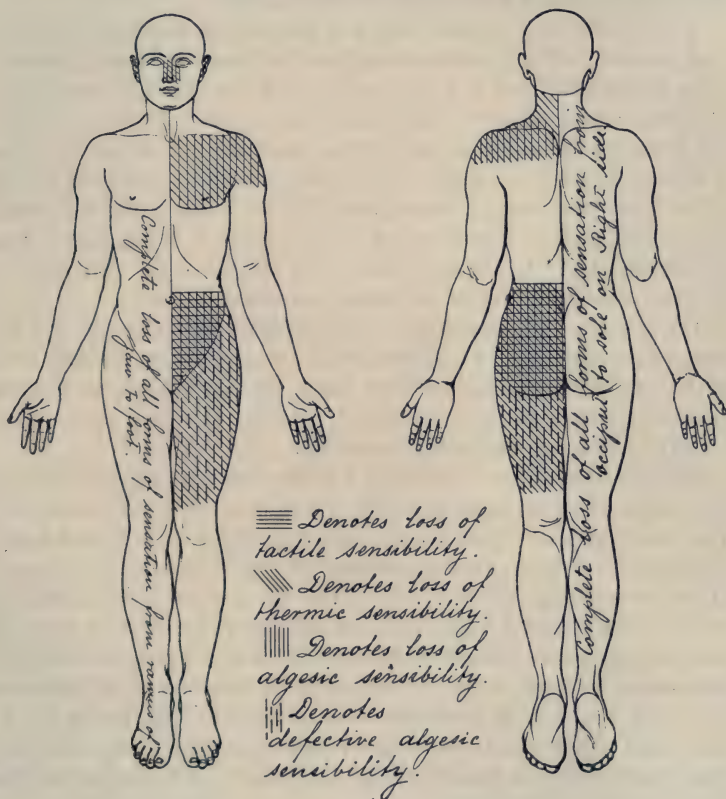


CHART 3.

responding muscles on the right side. The right knee-jerk is active, the left very exaggerated; there is well-marked left ankle-clonus, but only a slight tendency to right ankle-clonus. The plantar reflexes are normal. There is sometimes considerable difficulty in micturition, but never incontinence. Constipation is very obstinate.

CONDITION OF SENSATION.—*Subjectively*, a burning sensation of the sole of the right foot is very persistent. *Objectively*, see Chart 3.

On the head and face, sensation of all forms is quite normal, except that there is complete loss of thermic and algesic sensibility with retention of tactile sensibility over the nose and ears.

On the right half of the neck, trunk and right limbs, there is complete loss of all forms of sensibility, including loss of muscular sense, and sense of position in the right arm.

On the left half of the body the affection of sensation is more complicated. Thus, *on the neck*, there is loss of thermic sensibility posteriorly, but not anteriorly. *On the trunk*, there is loss of thermic and algesic sensibility over the shoulder, anteriorly and posteriorly, and over the front of the chest from the clavicle to a little below the nipple. There is loss of all forms of sensibility over the whole lower part of the trunk anteriorly and posteriorly, below the level of a line lying a little above the umbilicus. Over the rest of the trunk, and over the left side of the genitals, sensibility of all forms is normal.

On the left arm sensation is unaffected; while *on the left leg* there is loss of thermic and diminution of algesic sensibility over the thigh and leg as far down as the upper border of the patella anteriorly, and the upper extremity of the popliteal space posteriorly. The rest of the leg is normal.

TROPHIC EFFECTS.—There is no obvious wasting of muscles anywhere, no cyanosis of the skin, no hyperidrosis. On exertion or after injection of pilocarpine, only the right half of the body perspires. On the exterior surface of the proximal phalangeal articulation of the left ring finger, and on the interior surface of the distal phalangeal articulation of the right ring finger are small recurring growths. Each commences as a small roughness of the skin, which increases till it reaches about the size of a sixpence. Generally it cracks at some period of its growth so that a hard, dry, rough elevation is formed, which gradually heals entirely, but recurs later in exactly the same spot. No similar marks have been noted elsewhere.

The spine shows no prominence of, or tenderness over, any part. The function of all cranial nerves is normal. The right pupil is larger than the left. Lungs, heart, digestive and urinary systems are normal.

September 15.—Patient burnt two large blisters on the outside of the right forearm without feeling any pain.

November 15.—Patient's general condition remains unchanged. Extension of the head is now very feeble, and all movements of the left arm are very weak, especially abduction of the shoulder.

The right arm shows no loss of power, but when the arms are extended, athetoid movements are observed in the fingers of the right hand; while they occur very slightly, and only occasionally in the fingers of the left hand. All the muscles of the arm react to faradism, but those of the right arm rather more readily than those of the left. The left leg has become weaker and is dragged more in walking. The knee-jerks are much exaggerated and

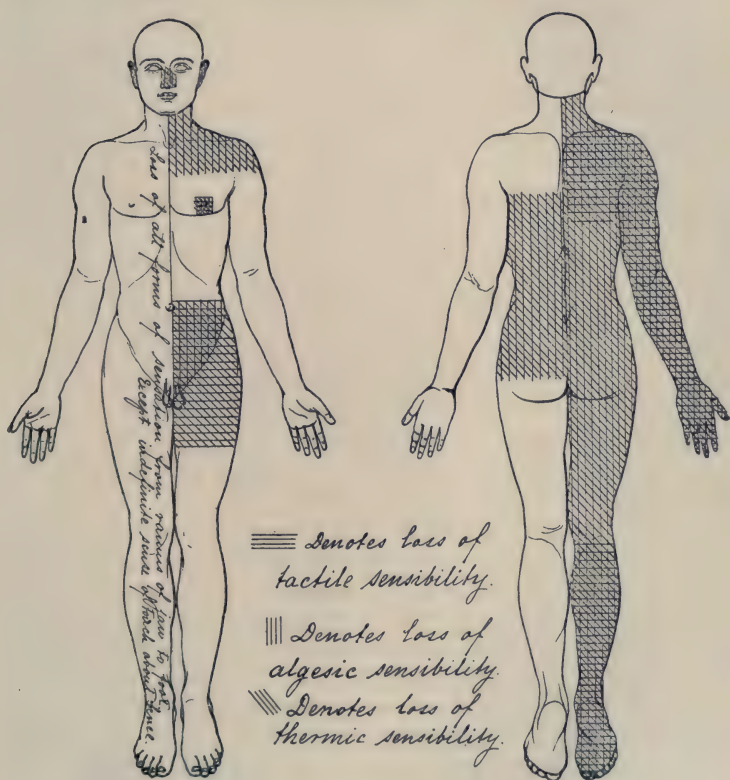


CHART 4.

equal. There is double ankle-clonus, perhaps rather more marked on the left side. The right plantar reflex is greater than the left. The right pupil is still larger than the left; otherwise, the condition of the pupil and ocular movements are normal. The left side of the face acts rather more strongly than the right, and the tongue is protruded slightly to the right. Very obstinate constipation and occasional difficulty in micturition (but never

incontinence) are still present. Swallowing is quite normal. There is no tenderness over the spine.

The *condition of sensation* on this date is given on Chart No. 4.

HEAD AND FACE.—Sensibility remains as before, *i.e.*, normal, except for loss of thermic and algesic sensibility over the nose and ears.

RIGHT SIDE OF BODY.—*Anteriorly*, there is loss of all kinds of sensibility over the whole of the right half of the body below the ramus of the jaw, except that at or about the knee there is an indefinite sense of touch.

Posteriorly, there is loss of algesic and thermic sensibility all over, from occiput to sole, and loss of tactile sensibility over the whole of the arm, the scapular region, and the leg beneath the knee. Elsewhere, tactile sensibility is normal.

There is complete loss of muscular sense and sense of position in the right arm.

LEFT SIDE OF BODY.—*Anteriorly*, there is loss of algesic and thermic sensibility over the neck, shoulder, and chest down to midway between the clavicle and nipple. From this level to the umbilicus, sensibility is unaffected, except over a small area around the nipple, which has lost all forms of sensibility.

The abdomen, below the umbilicus, has lost all forms of sensibility, but the genitals are unaffected.

The upper half of the thigh has lost tactile and thermic sensibility; the rest of the leg is unaffected.

Posteriorly, tactile sensibility is normal all over, but there is loss of algesic and thermic sensibility from a little above the lower angle of the scapula down to the gluteal region.

January 7, 1893.—Radical cure of hernia performed to-day. Patient began to be troubled with a cough; his breathing got worse, and he died on January 10.

The autopsy showed nothing abnormal in the lungs or anywhere else, except in the spinal cord.

The *microscopical examination* of the cord was imperfectly performed, owing to a breakdown in the method of hardening. At the level of the fourth cervical nerves (fig. 11), the anterior parts of both posterior columns are occupied by an irregularly-shaped cavity, surrounded by a neuroglia-like tissue. There is no evidence of a definite lining membrane to the cavity. The neuroglial tissue is seen to pass posteriorly as a finger-like process, between the columns of Goll and Burdach on either side, both these columns showing secondary degeneration, especially Goll's column.

The new tissue reaches the neck of both posterior horns, and in the left posterior horn there lies a cavity surrounded by neuroglial tissue, this cavity being continuous with the larger cavity in the posterior columns already mentioned. There is no appearance of a central canal, but the anterior commissure is intact. The vessels inside the cord, and in the pia-arachnoid, are large, congested, and distended with red blood corpuscles.

Besides the degeneration already noted in the posterior columns, there is almost complete degeneration of the left crossed pyramidal tract; and the degeneration is continued forward in the position of the antero-lateral descending tract, as far as the antero-median fissure. There is no degeneration in the antero-lateral region of the right side.

At the level of the twelfth dorsal nerves (fig. 12) there is seen to be a well-marked central canal, and from the posterior surface of the grey commissure there extends backwards a wedge-shaped area of neuroglia-like tissue into the postero-median septum. In some of the sections at this level, a few normally stained nerve-fibres are observed cutting off the central-canal region from the wedge-shaped area of new growth. The cavity in the left posterior horn still persists, but it is now quite cut off from the region of the central canal; it is surrounded by a fair amount of neuroglia-like tissue. At this level, there exists the same marked degeneration of the left crossed pyramidal tract, extending forward to a slighter extent into the antero-lateral region.

REMARKS.

In this case, as in Cases 1 and 2, the new growth and cavity formation involve the grey commissure, the intermediate grey matter and the anterior parts of both posterior columns; but here only one posterior horn—viz., the left—is implicated, and the secondary descending degeneration is limited to the left side. This preponderance of pathological affection of the left side accords with the clinical facts that the paresis was almost limited to the limbs of the left side, there being only slight weakness of the right leg, while the affection of all forms of sensation was greatest on the right side, and the sense of touch was implicated almost exclusively on that side, *i.e.*, clinically it is an example of Brown-Séquard's palsy.

In the midcervical region, the communication of the

cavity in the posterior horn with the cavity in the posterior columns is very distinct, and there is certainly no trace of an endothelial lining to any part of the cavity.

The cavity in the left posterior horn in the lower dorsal region is probably continuous with the cavity in the corresponding situation in the midcervical region, but the absence of a continuous series of sections renders this uncertain.

The extension of the new-growth in the midcervical region along the septum between Goll's and Burdach's column on either side, and the limitation of the gliosis in the lower dorsal region to a wedge-shaped area in the front part of the postero-median fissure are examples of implication of two favourite sites for syringomyelic changes (Schlesinger⁵).

SOME GENERAL REMARKS ON THE CONDUCTION OF SENSORY IMPRESSIONS IN THE SPINAL CORD.

It has been assumed in many cases that the sensory symptoms of syringomyelia are due to the interruption of the afferent conducting tracts in the spinal cord; but, as Dr. Ferrier pointed out, when the above recorded cases were exhibited before the Neurological Society, the anæsthesia was mainly segmental in distribution, sensation not being affected in the parts below the lesion—except in one instance (Case 3).

Hence these cases show that the pathological changes occurring in syringomyelia may produce disturbances of sensation in one of two ways:—

(a) By interfering with incoming sensory impressions through one or more posterior roots—segmental anæsthesia.

(b) By severing the continuity of the conducting paths, thus abolishing sensation in the parts of the body below the lesion.

We find examples of both conditions in the cases here recorded. Thus, in Cases 1 and 2, throughout the whole course of the disease, the defects of sensation were limited to the spinal segments corresponding roughly to the pectoral

⁵ *Schlesinger*, "Die Syringomyelie," 1895, pp. 159-162, and fig. 14.

girdle, and illustrating the segmental nature of the anæsthesia (*vide* charts 1 and 2). In Case 3 both varieties of sensory disturbance were observed ; on one side a segmental anæsthesia, corresponding to certain posterior nerve-roots ; on the other, total anæsthesia below the lesion from interference with the afferent paths (*vide* charts 3 and 4).

This condition is what might be expected from the *post-mortem* examination of the spinal cords, already detailed and figured. Here we note that two individual parts of this structure are seats of election of new growths :—

(a) The posterior root-zones, and adjacent parts of the postero-median columns.

(b) The gelatinous substance of Rolando and adjacent parts of the posterior horn.

Interference with the former structures, more especially the posterior root-zone, is in all likelihood, capable of effecting the segmentary sensory disturbances ; while the anæsthesia below the seat of the lesion is due, in our opinion, to destruction of the posterior horn and gelatinous substance of Rolando. Case 3 is highly instructive in that both conditions were observed. On one side—the right—the new growth is limited to the anterior portions of the posterior root-zone and adjacent part of the postero-median column (*fig.* 11). The sensory defects arising from this lesion are segmental in nature, and are upon the same side of the body, but, as shown in charts 3 and 4, they are included in and obscured by the anæsthesia from lesion of the conducting tracts on the opposite side. On the other side—the left—there was a lesion involving the posterior horn and gelatinous substance as well as the posterior root-zone and adjacent part of the postero-median column, in the upper cervical region of the spinal cord, as a result of which all forms of sensation were abolished on the right side below this level ; while there was segmental anæsthesia on the left side of the body (charts 3 and 4). As there existed on the side of the lesion much weakness of the arm and leg, with descending secondary degeneration in the pyramidal tracts, there was presented the classical symptomatology of the so-called Brown-Séquard paralysis. In a previous number of

this Journal (BRAIN, pts. 70 & 71, p. 231) it was shown that experimental destruction of the tubercle of Rolando and the adjacent part of the posterior horn in monkeys was productive of a constant series of objective sensory phenomena, consisting mainly of abolition of all forms of sensation over the area of trigeminal distribution on the side of the lesion, and loss of response to painful impressions on the opposite side, the response to ordinary tactile impressions being temporarily abolished on the side of the lesion. The conclusion drawn from these experiments was, that the gelatinous and spongy portions of the posterior horns were of prime importance in the conduction of sensory impressions at all events in the upper portions of the spinal cord, more especially in the neighbourhood of the spinal-medullary junction.

The large number of experimental observations made within recent years upon hemisection of the spinal cord by Mott, Marshall,⁶ and others, tend to show that there is no constant distribution of the sensory fibres, which enter by the posterior nerve roots. The cases of unilateral lesion of the spinal cord in man, on the other hand, show a much more constant relation between the several forms of cutaneous sensibility. Reference to a large number of cases of the so-called Brown-Séquard paralysis, scattered throughout medical literature, has shown that the sense of pain is invariably abolished on the side opposite the lesion in association with the sense of temperature, while these are not uncommonly dissociated from tactile sensibility. Whether the sense of touch is abolished along with the other forms of cutaneous sensation, or not, would appear to depend upon the extent of destruction of the conducting paths, as is presently to be shown.

And this leads us to the consideration of one of the most interesting features brought out by a study of these cases, viz., the curious dissociation which exists between the several forms of cutaneous sensation. In Cases 1 and 2 loss of thermic sensibility was first observed. In Case 1 there

⁶ *Proc. Roy. Soc.*, 1895, p. 475.

existed throughout the whole course of the disease a dissociation between thermic sensibility on the one hand, and tactile and painful sensibilities on the other. But in Case 2 there occurred a progressive interference, with eventual abolition, of the sensibility, first of temperature, then of pain, and lastly of touch.

These facts do not necessarily indicate the existence of separate tracts in the spinal cord for the conduction of the several forms of cutaneous sensation. As the order in which they are lost appears to be constant, it is difficult to believe that disease should invariably at the outset implicate tracts subserving thermic sensibility, and later on those of pain and touch. This may be explained on the hypothesis *that the more fundamental the form of sensation, the more extensive is the provision for its conduction in the spinal cord.*

Hence in the early stages, when the lesion is small, thermic sensibility only is impaired; while in the late stages, when the destruction of tissue is presumably greater, the less organised forms of sensation are impaired or abolished; and, conversely, after a spinal lesion, which has at the outset abolished all forms of sensation, the appreciation of touch is that which first returns, while pain and temperature usually remain in abeyance. In amplification of this we have seen a monkey, in which both painful and tactile sensibilities were abolished by a lesion involving the posterior region of the spinal cord. Although the loss of pain persisted, the sense of touch returned on the side which was least involved, a circumstance which may find explanation in the more widespread distribution throughout the posterior horn of the fibres subserving tactile sensibility. (BRAIN, *loc. cit.*, p. 238).

To look more particularly at the individual cases, it is clear that this generalisation holds good, whether the lesion involves the conducting or the segmental arrangements. Thus, in cases where the posterior root zones were only or chiefly implicated, loss of thermic sensibility was that first affected, and in Case 1 this was the only form of cutaneous sensibility abolished.

This view harmonises with what must undoubtedly occur,

both in the peripheral nerves and in the end-organs in the skin. It is as difficult to realise the existence of separate end-organs, or of separate nerve fibres,⁷ for the reception and conduction of the several forms of cutaneous impressions, as it is to assume separate paths for their transmission in the spinal cord, or separate centres for their perception in the cerebral cortex.

EXPLANATION OF THE PLATES.

Figs. 1—5 represent the appearance of the Spinal Cord in Case I.

- FIG. 1.—Mid-cervical region.
- FIG. 2.—Lower cervical region.
- FIG. 3.—Upper thoracic region.
- FIG. 4.—Lower thoracic region.
- FIG. 5.—Lumbar enlargement.

Figs. 6—10 are taken from Case II.

- FIG. 6.—At level of second and third cervical nerves.
- FIG. 7.—At level of fifth cervical nerves.
- FIG. 8.—At level of sixth and seventh nerves.
- FIG. 9.—Neuroglioma ganglionare (Eosin and Logwood).
- FIG. 10.—Central grey matter (Marchi's method).

Figs. 11 and 12 depict the Spinal Cord of Case III.

- FIG. 11.—At the level of the fourth cervical nerves.
- FIG. 12.—At the level of the twelfth dorsal nerves.

Detailed descriptions of the figures are given in the text.

⁷ In some interesting experiments by Waller, thermic sensibility was the last to return after compression of the trunk of the radial nerve (referred to by Mitchell. "Remote consequences of injuries to nerves," 1895, p. 21).



Fig. 1.

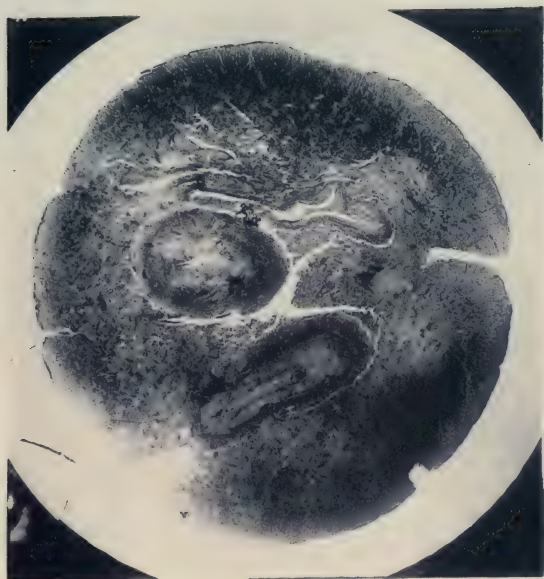


Fig. 2.

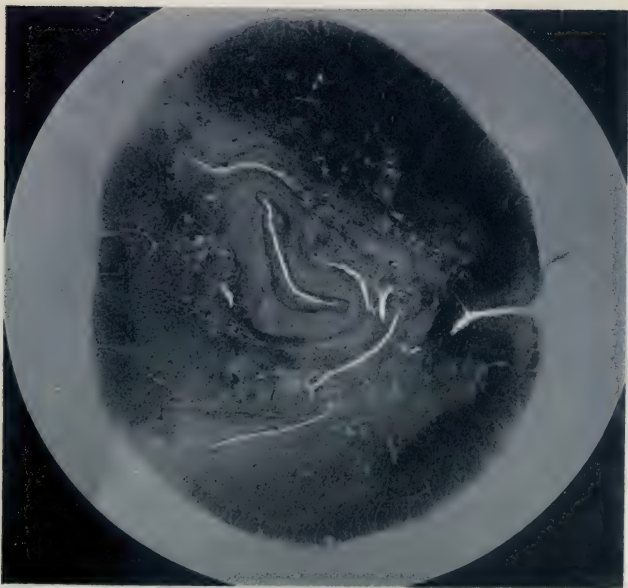


Fig. 3.

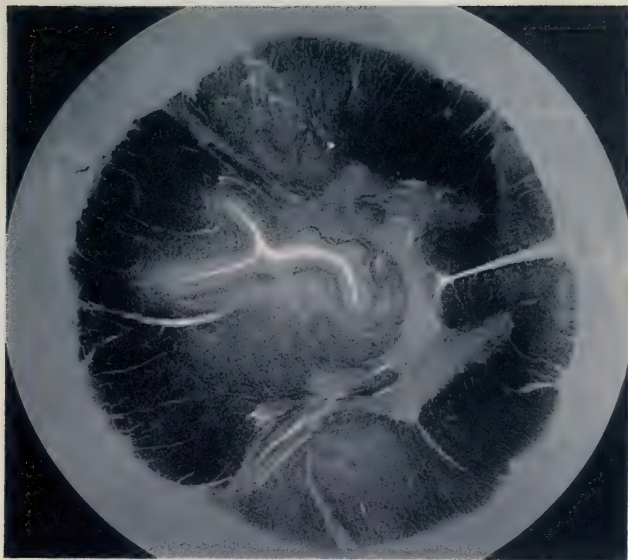


Fig. 4.



Fig. 5.

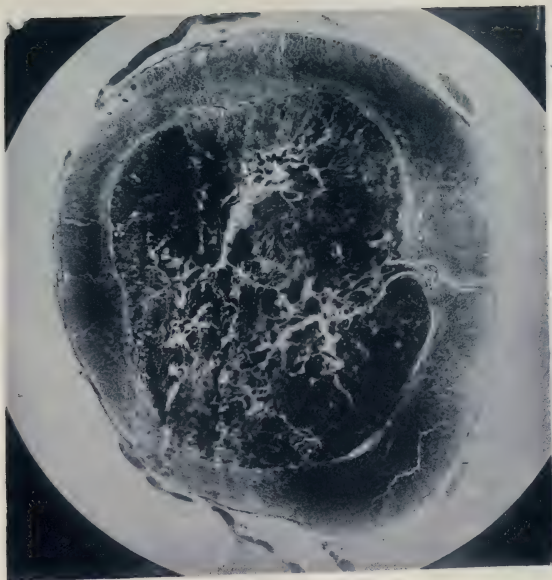


Fig. 6.

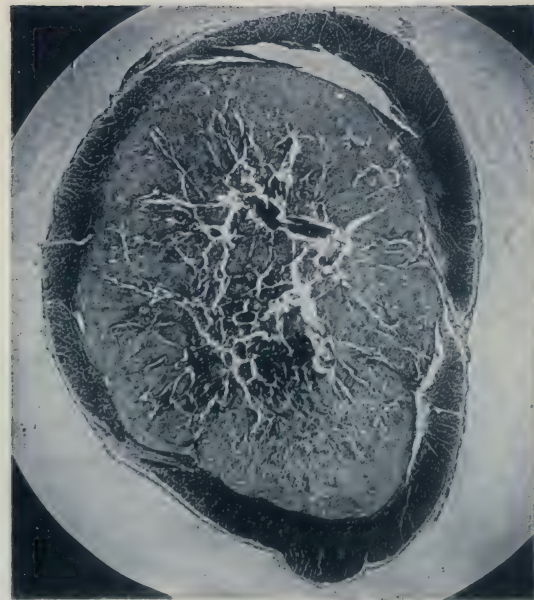


Fig. 7.

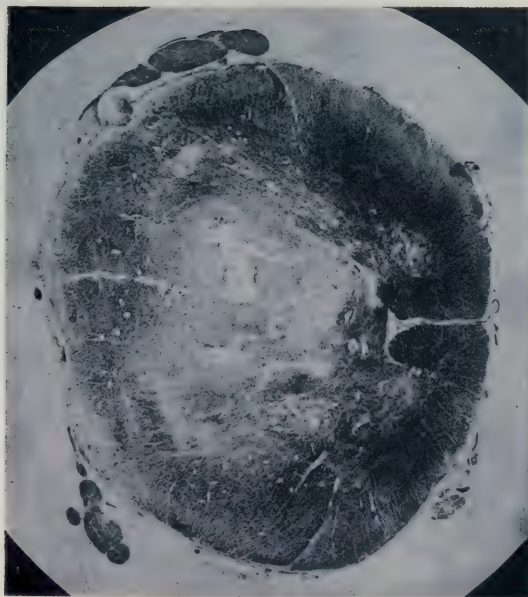


Fig. 8.

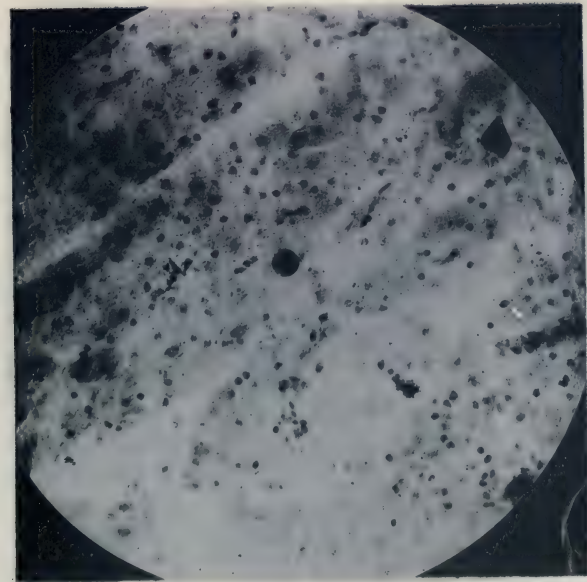


Fig. 9.

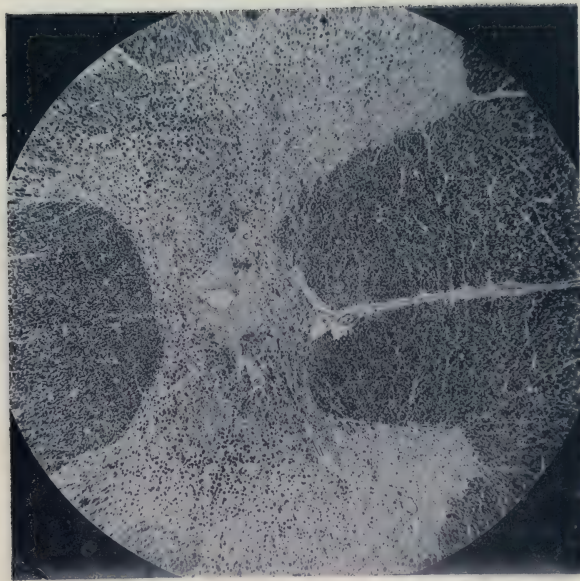


Fig. 10.

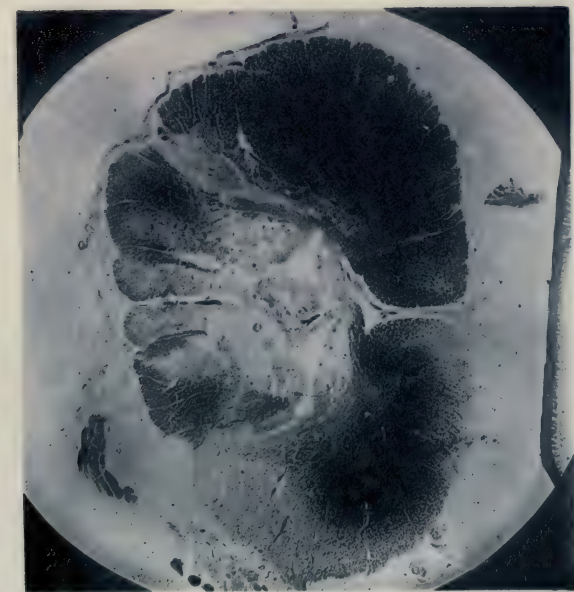


Fig. 11.

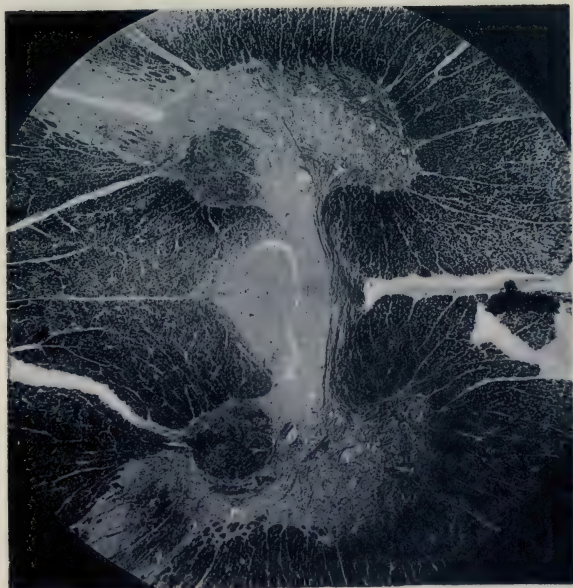


Fig 12.

ON A DESCENDING DEGENERATION IN THE POSTERIOR COLUMNS IN THE LUMBO-SACRAL REGION OF THE SPINAL CORD.

BY ALEXANDER BRUCE, M.A., M.D., F.R.C.P.E.

*Assistant Physician to the Royal Infirmary; Lecturer on Pathology at
Surgeons' Hall, Edinburgh;*

AND

ROBERT MUIR, M.A., M.D., F.R.C.P.E.

*Pathologist to the Royal Infirmary, and Assistant to the Professor of Pathology,
University of Edinburgh.*

IN 1867, Bastian (1) figured a descending tract in the posterior columns of the cord, which had degenerated subsequently to an injury in the upper part of the cervical enlargement.

Thirteen years later (1880), Kahler (1) and Pick (2) described a somewhat diffuse descending degeneration in the posterior columns after fracture of the sixth cervical vertebra. In the same year, Strümpell (3) described and figured a small area of descending degeneration in the dorsal region, following a transverse myelitis. This area was situated at the junction of the postero-median and postero-external columns, near the posterior commissure. Westphal (4) also figured and described a downward degeneration of "two narrow bands parallel to the posterior horns" as being present at the ninth dorsal root, in a case in which there was a tumour at the fourth and fifth dorsal segments. His figure shows the degenerated area to be in the postero-lateral column, but not quite reaching either the posterior commissure or the periphery of the cord.

In 1883, Schultze (5), in a case of tumour at the level of the fourth and fifth cervical roots, found a degenerated tract in each postero-external column, which descended for about $2\frac{1}{2}$ centimetres (nearly one inch). He described and figured it as beginning some distance from the posterior commissure, and terminating some little way from the periphery, and as being parallel to the inner margin of the posterior cornu. He termed it, from its form, the "comma-shaped" tract, which name it still retains. It is quite evident that this tract is the same as that described by Westphal. Schultze regards it as being formed of descending branches of the posterior root fibres, but without adducing any adequate reason for his opinion.

Tooth (6), in 1889, described three cases in which this tract was affected. He found that the degeneration of the comma-shaped tract extended as low as the eighth dorsal segment, in a case in which the cord was crushed between the eighth cervical and first dorsal roots; in another case, in which the cord was crushed between the sixth and seven cervical roots, the degeneration extended to the sixth dorsal segment; and, in the third case, a fracture dislocation of the fifth dorsal vertebra, the degeneration extended an inch below the lesion. He also found that the tract degenerated downwards on the same side, after hemi-section of the cord, and considers that the degeneration is probably more distinct after lesions above the fifth dorsal root than below that level. He failed to find any degeneration in this tract after section of the roots only, and, therefore, regards it as being probably commissural in nature.

Schmaus (7), in 1890, described and figured an area of descending degeneration in the lower cervical and upper dorsal cord, which closely corresponds in form and position with the comma-shaped tract, as described by Schultze and Tooth. The degeneration was due to a softening in the cervical enlargement, following an injury resulting from a fall.

Pfeiffer (8), in 1891, in a case of sarcoma, involving the three lower cervical and first dorsal segments, found at the level of the middle and lower dorsal roots, a degeneration involving part of the postero-external, and the adjacent portion

of the postero-median columns. In the absence of figures one cannot form any more precise idea of the position and shape of the degenerated tract in this case. It did not, however, extend below the lowest dorsal segment.

Bruns (9), in 1883, figures a descending tract in the postero-external columns, which degenerated as far as the fifth dorsal segment, after a lesion at the junction of the cervical and dorsal regions. He terms it the "comma-tract," but his figures show a somewhat more extensive degeneration than is indicated by Westphal, Schultze, and Tooth.

Gombault and Philippe (10), in a case of gradual compression by a tumour, at the third cervical segment, found a descending degeneration in a narrow band in the posterior columns, situated nearer Goll's column than the posterior horn, but not reaching either the commissure, or the periphery. It extended downward nearly to the sixth cervical segment. They agree with Tooth in thinking that the "comma tract of Schultze" is not directly connected with the descending fibres of the posterior nerve-roots, since they found it undegenerated in a case (of lead poisoning) in which the fifth cervical root was destroyed.

These cases have established beyond doubt (*a*) that in the cervical and dorsal portions of the cord, as low down at least as the ninth dorsal segment, there is in the postero-external column a tract, which degenerates in a descending direction; (*b*) that this tract is situated in the position first described by Schultze, namely, about the middle of the breadth of the postero-external column, parallel to the inner margin of the post cornu, and not quite reaching either the posterior commissure or the periphery of the cord; (*c*) that the degeneration of this tract passes downwards for only a limited distance. It is as yet uncertain what this distance is, how the tract begins, or how it terminates. Further research is required to settle these points; but it seems clear that the tract is not found in the lumbar region in the same relation to the posterior cornu, and that, if it exists there at all, it must take the form of a diffuse degeneration, or occupy some other position. None of the above-mentioned observers, however, with the

exception of Gombault and Philippe, appear to have found any degeneration in this region. Daxenberger (12) describes a descending degeneration, extending from the first dorsal segment, as far downward as the upper lumbar region, in both the postero-external and postero-median columns. The degeneration in the former was comma-shaped, and was present in the upper and middle dorsal regions. In the postero-median column, the degeneration lay close along each side of the posterior median septum, in its anterior half, but not reaching quite to the commissure; and was present throughout the dorsal and upper lumbar regions. This degeneration in the postero-median column has not been found in any other case, and it seems difficult to reconcile it with the facts already mentioned, or with the fact that ascending degeneration of this column leaves no unaffected area, as it ought to do, if the tract in the cervical and dorsal regions contained descending fibres.

We have recently, however, had an opportunity of examining a case which appears to us to establish definitely the existence of a descending tract in this region. In a case of fracture-dislocation of the twelfth dorsal vertebra, the cord was completely crushed and destroyed in the upper lumbar region, over a length of about one inch, death taking place within five weeks of the injury—at a time, therefore, specially favourable for ascertaining the degeneration by Marchi's method. The cord was hardened for four weeks, in the usual way, in Müller's fluid, and then small pieces at various levels, above and below the lesion, were transferred for fourteen days to Marchi's fluid.

In sections made from the lumbar and sacral regions, a diffuse degeneration was found throughout the whole of the antero-lateral columns. This degeneration was fairly uniformly distributed, except in the position of the crossed pyramidal tracts, where it was rather more marked, and in the anterior columns, along the margins of the anterior median fissure, and the adjacent portion of the anterior surface of the cord (the *descending sulco-marginal tract of Marie*). These degenerations diminished in a descending direction.

In the posterior columns there were various degenerations. Immediately below the spot at which the cord had been destroyed, these columns were degenerated in an irregular manner, the degeneration being manifestly dependent upon the direct injury to the fibres. It should further be noted that several of the posterior roots in the upper lumbar region, not extending below the fourth lumbar segment, had been injured between the cord and the posterior root ganglion, but that the roots below this level were quite intact.

At the level of the third lumbar segment there was a somewhat dense degeneration in the anterior root zone of Flechsig. This was undoubtedly, for the most part, due to the degenerated fibres from the posterior roots, which could be seen entering the zone from behind and leaving it in front, to pass into the grey matter.

A second area of degeneration was found on either side closely applied to the hinder third of the posterior-median septum, and along the inner half of the periphery of the posterior columns. The breadth of these areas, as is shown in the diagram (fig. 1), was somewhat greater along the periphery than along the posterior median septum, where, on the other hand, the degenerated fibres were more closely aggregated.

Thirdly, there was a slight diffuse degeneration in the middle root zone. The part of the posterior columns which lay next the middle third of the septum, between the periphery and the posterior commissure, shewed almost no degeneration.

At the level of the fourth lumbar segment (fig. 2.) the degeneration along the inner margin of the posterior cornua had become much less marked, evidently because at this level the degenerated root fibres of the injured nerves had almost entirely ceased to enter the cord. The second area of degeneration, namely, that along the posterior median septum and the adjacent portion of the periphery of the cord, had greatly increased in extent. There was now a narrow band of degenerated fibres, extending along the whole length of the septum, from the posterior commissure to the

the periphery. The fibres were more closely aggregated, and the band was rather thicker at the posterior portion of the septum than in the anterior two-thirds. On the other hand, the degeneration at the periphery did not extend quite so far in a lateral direction as at a higher level. In the middle root zone there were practically no degenerated fibres.

In the upper sacral regions (figs. 3 and 4), the median degeneration had assumed a distinctly triangular form posteriorly, while still extending forward as a narrow band on either side of the septum as far as the posterior commissure, and apparently extending into the grey matter. At the junction of the lumbar and sacral segments the degenerated area had apparently attained its maximum. In the lower sacral and coccygeal areas it gradually diminished in size, while retaining the same defined wedge-shape (figs. 5 and 8). In the three lower sections figured the degenerated fibres could be traced into the bases of the posterior cornua at either side of the central canal.

There is no doubt whatever that this degeneration is a descending one. There were no injured roots below the level of the fourth lumbar segment which could possibly have given rise to it, and there was no other injury of the cord at its lower end. It would appear most probable that the tract increases in size by the gradual passage into it of degenerated fibres from other regions of the posterior column at higher levels, but we are unable to determine directly whether the tract is formed from descending fibres of the posterior roots in the lumbar region, or within the cord itself. This point might be settled by cases of section of, or tumours within, the cord on the one hand, or, on the other, by affections of the nerves produced by section or destruction of the cauda equina, or by disease, such as locomotor ataxia, which leads to degeneration of the root fibres within the cord. If the fibres are of endogenous origin, that is, if they originate within the cord, they would degenerate after lesions within the cord, and would be spared in injury or lesion, involving the roots only.

Barbacci (11), in a case of neoplasm in the sixth dorsal segment, found a descending degeneration in the posterior

columns, which at the level of the eighth dorsal segment was diffused over all the areas, but was most marked in the portion of the postero-external column next the posterior cornu. At the level of the last dorsal and first lumbar segments, this area tended to become grouped more towards the inner part of the postero-external column, and at a still lower level appeared as a narrow band along the posterior half of the posterior median septum. This case is referred to by Marie ("Lectures on Diseases of the Spinal Cord," Sydenham Society's translation, p. 38). Barbacci also found that, after section of the cord of the cat at the second and third lumbar segments, there were a few degenerated fibres diffusely scattered throughout the whole posterior column. In another case, after hemi-section of the cord of a dog between the tenth and eleventh dorsal roots, there was at the lower dorsal region, a descending degeneration in the posterior columns which formed an irregularly triangular area near the hinder part of the septum. There were also some degenerated fibres scattered throughout the rest of the column. At the level of the lumbar enlargement only a few degenerated fibres remained, situated mostly in the anterior portion of the column.

Gombault and Philippe (*loc. cit.*) in a case of total transverse myelitis in the upper part of the lumbar enlargement, found below the lesion, on either side of the posterior median septum, a triangular area of degeneration, which on the right side almost reached to the posterior commissure. At lower levels this triangle receded gradually from the posterior commissure, and ultimately its apex lay midway between the commissure and the periphery of the cord.

On the other hand, in two cases described by Schultze, in one of which the cauda equina had been crushed, and in the other the roots of the sciatic nerve and the lumbar portion of the cord had been destroyed, a small triangle of undegenerated fibres was found above the lesion at the junction of the posterior median septum with the periphery of the cord. The rest of the posterior columns, with the exception of the cornu-commissural tract of Marie, was degenerated.

Gombault and Philippe further describe a case of loco-

motor ataxia in which the fourth and fifth sacral roots on both sides, and the sixth sacral (coccygeal?) root on the right side, were destroyed. Here they found a tract of undegenerated fibres in relation to the posterior median septum. This tract, at the level of the fifth and sixth sacral roots, was small and of a triangular form. Above this level it passed further forwards along the posterior median septum, assuming, at the same time, an oval form. At the level of the fourth lumbar it became reduced in size. The authors give satisfactory reasons for concluding that these fibres are not derived from the coccygeal root, but must belong to a descending tract of the cord.

The persistence of similar areas of healthy fibres in the lower lumbar and sacral regions in locomotor ataxia is well known; but the reasons given why they fail to degenerate along with the adjacent portions of the posterior columns have hitherto been unsatisfactory. If they represent, as we now believe they do, fibres descending from higher levels of the cord, it is quite intelligible why they have escaped the degeneration which has overtaken those fibres which are a continuation of the posterior roots within the cord.

Hoche (13) describes two cases of compression of the cord, one at the eighth cervical, and the other between the fourth and sixth dorsal roots, of three and six weeks standing respectively, in which the degenerations in the posterior columns in the dorsal, lumbar, and sacral regions presented closely similar appearances. He found that after a lesion in the cervical region, a descending degeneration could be traced as far as the lowest portion of the *conus terminalis*. The fibres of the comma-shaped tract ended above the first appearance of the "oval field of Flechsig" (*i.e.*, above the twelfth dorsal root), by passing into the grey matter, and thus took no share in forming the "oval field." In the lumbo-sacral region there was a degenerated area on either side of the posterior median septum, which, in the middle and lower part of the lumbar enlargement, did not quite reach the posterior commissure or the periphery, but in the sacral region extended back to the periphery, and also sent its fibres forward into the grey matter. The lowest fibres of the degen-

eration ended in the *conus terminalis* by bending forward into the grey substance. This tract, at the level of the twelfth dorsal root, occupied a small area extending along the periphery of the cord from the posterior septum about half way to the entry of the posterior roots. In the upper dorsal region it was further outwards and more diffuse, forming a wedge-shaped area in the postero-external column, with its base at the periphery, and its apex touching the tail of the comma tract. This relation existed as far down as the ninth dorsal segment.

According to Hoche, therefore, the descending tract in the posterior columns in the lumbo-sacral region, and the comma-shaped tract of Schultze, both originate in the cervical part of the cord; but they are quite independent of each other, and the lumbo-sacral tract does not represent a downward continuation of the comma-shaped tract.

CONCLUSIONS.

It seems *proved* :—

(1) That there is a descending tract in the posterior columns in the lumbo-sacral region.

(2) That this tract in the upper lumbar region is somewhat irregularly diffused through the greater part of the column.

(3) At the level of the third lumbar root (fig. 1) it becomes collected along the margin of the hinder portion of the posterior-median-septum and the periphery of the inner half of the posterior columns.

(4) That at the level of the fourth lumbar root (fig. 2) it extends forward as a narrow band along the side of the septum as far as the posterior commissure. Below this level, at the first and second sacral roots (figs. 3 and 4) the tract has the form of a narrow wedge, with its base at the periphery, and its apex at the posterior commissure. Below the third sacral root (fig. 5) the tract diminishes in size as far as the coccygeal segment (figs. 6, 7 and 8), while retaining its triangular form, and its relation to the posterior median septum.



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.

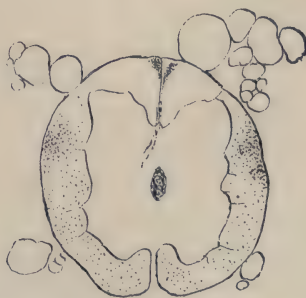


FIG. 6.

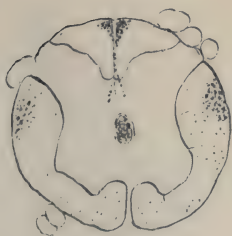


FIG. 7.

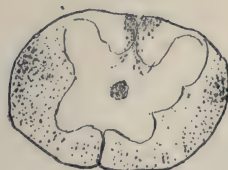


FIG. 8.

(5) That its fibres terminate by passing forwards into the grey matter at the base of the posterior horn of the same side (figs. 6, 7 and 8).

It seems *probable* :—

(1) That the tract is not directly continuous with the fibres of the posterior roots in the lumbo-sacral region. This seems indicated by its escape in locomotor ataxia, and in cases of destruction of the cauda equina.

(2) That it is also independent of the “comma-shaped” tract. This seems indicated by the failure of any observer to trace the latter as a distinct tract to a lower level than the ninth dorsal segment.

(3) That it is independent of the cornu-commissural tract of Marie. This tract seems to have entirely escaped degeneration in our case, a fact which seems to indicate that if its fibres have a descending direction, they must originate below the level of the lesion, in the upper lumbar region.

(4) That the fibres which compose this tract probably enter it at different levels.

(5) That it appears to correspond in position to the “oval field” of Flechsig, and the “dorso-median sacral bundle” of Obersteiner (14).

NOTE.—As the tract bears a relation to the *posterior median septum*, analogous to that borne by the *sulco-marginal tract* of Marie to the *anterior median fissure* or *sulcus*, we would suggest that it may be termed the *descending septo-marginal tract*. This name would indicate briefly its course, its position in the posterior columns, and its relation to the septum.

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EXPLANATION OF PLATES.

- FIGURE 1. T. S. of spinal cord at the level of the third lumbar root.
- „ 2. T. S. of spinal cord at the level of the fourth lumbar root.
- „ 3 & 4. T. S. of spinal cord at the level of the first and second sacral roots.
- „ 5 & 6. T. S. of spinal cord at the level of the third and fourth sacral roots.
- „ 7 & 8. T. S. of spinal cord at the level of the fifth sacral and coccygeal roots.

ON THE SYMPTOMATOLOGY OF GROSS LESIONS (TUMOURS AND ABSCESES) INVOLVING THE PRÆ-FRONTAL REGION OF THE BRAIN.

BY R. T. WILLIAMSON, M.D.LOND., M.R.C.P.

*Medical Registrar Manchester Royal Infirmary, and Assistant in Medicine
Owens College.*

THE symptoms produced by tumours and abscesses involving the præ-frontal region of the brain are often somewhat ill-defined, and probably mistakes in localisation occur more frequently in lesions of this region than in lesions of any other part of the brain. In many cases, however, a careful study of all the symptoms will indicate the seat of the disease.

During the last ten years five cases of tumour or abscess involving the præ-frontal region have been admitted into the medical wards of the Manchester Royal Infirmary, and an analysis of the symptoms in these cases, and in forty-five other cases, reported in medical literature during recent years, presents several points of interest.

Experiments on animals have shown that lesions of the præ-frontal region are not followed by such well-defined symptoms as lesions of other parts of the brain. Nevertheless, the results of these experiments are of great interest. They have been ably discussed by Professor L. Bianchi in the last volume of *BRAIN* (winter part, 1895, p. 497), and hence in this paper need only be briefly referred to. The views which have been advanced as to the functions of the præ-frontal lobe may be summarised as follows (Professor Bianchi):—

“(1) The præ-frontal lobe is the motor centre of the eyes and head on the opposite side, and in consequence of the

close relationship between the movements of these parts and attention, it is also the centre for attention (Ferrier).

“(2) It is the centre for the highest psychical functions. Destruction of it involves a real decadence of psychical activity (Wundt, Hitzig, Bianchi).

“(3) It is part of the so-called ‘Fühlsphäre,’ and as such is the motor centre of the dorsal muscles. Its highest development is not correlated with that of intellect, but with that of the dorsal musculature (Munk, Luciani).”

Professor Bianchi criticises the first and third views and records a number of experiments made on animals. From the psychical disturbance which he has observed after removal of the præ-frontal lobes he concludes, “that the frontal lobes are the seat of co-ordination and fusion of the in-coming and out-going products of the several sensory and motor areas of the cortex.” “The frontal lobes would thus sum up into series the products of the sensori-motor regions, as well as the emotive states which accompany all perceptions, the fusion of which constitutes what has been called the *psychical* tone of the individual.”

The following are abstracts of the notes of four cases of tumour and one of abscess, involving the præ-frontal lobes. To the kindness of Dr. Leech, Dr. Dreschfeld, Dr. Graham Steell, and Mr. Wright, I am indebted for the opportunity of reporting these cases.

Case 1.—Sarcoma of both præ-frontal lobes ; absent knee jerks ; unilateral optic neuritis ; spinal cord normal.—Mary O., aged 23 ; under the care of Mr. Wright and Dr. Dreschfeld. A sarcomatous tumour had been removed from the middle of the anterior part of the left thigh, but on November 20, 1893, the patient was re-admitted to the Manchester Royal Infirmary, owing to the appearance of a swelling about the size of an egg at the lower part of the cicatrix.

The patient, on admission, complained of a feeling of sickness. On November 23 she still complained of sickness, but had no pain anywhere. On the evening of the 26th she felt very sick and complained of headache on several occasions. On November 27, whilst having a bath, she had what appeared to be an attack of syncope. The pulse became very feeble, and the patient lost consciousness. Ether and brandy were given, but she remained

unconscious. The temperature was 97.6° . On November 28, she still remained unconscious, and the temperature rose to 100.8° in the morning, and 101.6° in the evening. I saw the patient for the first time on November 29. She then appeared to be unconscious. When the arms were raised into the vertical posture and allowed to fall, the right one fell down more suddenly than the left. When the right arm, leg, and right side of the face were pricked with a pin, no movement could be obtained, but slight movements were obtained on the left side. The pin prick appeared to cause pain on both sides. *The knee-jerk was absent on both sides*; plantar reflexes present; no ankle clonus. The abdominal and epigastric reflexes were present on the left side but not on the right. There was slight blurring of the margins of the right optic disc. Heart and lungs normal. November 30 there was slight optic neuritis in the right eye. The left disc was normal. The patient was in a semi-conscious condition, but could be roused by pinching or pricking with a pin. A few days later she would put out the tongue when asked to do so, but the knee-jerks remained absent on both sides. The optic neuritis in the right eye became more marked, but the left optic disc remained normal. On December 12 the difference between the limbs on the right and left sides was very slight. The patient could be made to perform slight movements of the right limbs, though she still remained in a drowsy state. No facial paresis could be detected. The tongue was protruded straight. She could not be made to answer any questions, but would occasionally speak to the nurse. *The knee-jerks were still both absent*. She could swallow food quite well. On December 27 she became quite comatose, and death occurred on the 28th.

The knee-jerks were carefully examined three or four days before death, but *they were both still absent*, and there was no ankle clonus. During the four weeks the patient was in the hospital the knee-jerks were always absent.

At the *post-mortem examination*, on the surface of the left cerebral hemisphere, in the præ-frontal region, was an oval tumour growth about 2 in. by $1\frac{1}{4}$ in., the long diameter corresponding to the position of the second left frontal convolution. On making vertical transverse sections of the brain, the growth was found to extend deeply (for about 2 inches at one spot) into the white matter of the left præ-frontal lobe. The tumour was very vascular, and at the centre was softened and broken down, giving rise to a cavity which contained yellow fluid and *débris*. In the white matter of the right præ-frontal lobe were two smaller

tumour growths, one just beneath the first frontal convolution, and one beneath the second convolution. The former was softened at the centre; both were vascular. Another section through the posterior part of the third frontal convolution showed the presence of a fourth tumour, situated in the middle of the white matter of the right præ-frontal lobe, and a very small growth invading just the anterior end of the right lateral ventricle.

The ascending frontal convolution, the motor region of the cortex, the motor fibres in the white matter and the basal ganglia were *not* invaded by tumour growth. In the white matter of the occipital lobe on the left side there was a small nodule of new growth about the size of a pea, and one in that of the right occipital lobe almost at the apex of the lobe.

The *cerebellum* medulla and pons were quite normal. The spinal cord was normal to the naked eye; after hardening in Müller's fluid it appeared normal on section. Pieces were taken from the lumbar, dorsal and cervical regions, and embedded in celloid, and sections made. Microscopically the cord appeared normal.

At the inner part of the left thigh, about the apex of Scarpa's triangle, was a tumour growth about the size of an egg. Microscopically, the cerebral tumour was found to be a very vascular sarcoma. There were no changes of importance in the other organs.

Case 2.—Abscess of right frontal lobe. Optic neuritis on right side only. Frontal and occipital headache. Absent knee-jerks.—William B.,¹ aged 19, admitted as an in-patient under the care of Dr. Steell, March 22, 1890. On February 18, 1890, the patient was attacked with influenza. The nasal discharge was profuse, and after recovering from the attack he continued to suffer from pain in the head, especially in the frontal and occipital regions (Previous to the attack of influenza he was not troubled with headache.) His mental condition became very dull and he was unable to return to his work. The pain in the frontal region became so severe that he was obliged to remain in bed. The mental condition became gradually more dull and stupid.

Condition on admission to the Hospital.—Patient is pale and badly nourished. His face has a dull, stupid expression. He takes very little notice of his surroundings, but always recognises his mother. He lies on his back in a drowsy condition, with his

¹ The complete history of this case has been published by the writer in the *Medical Chronicle*, 1890-91, vol. xiii., p. 423.

eyes half closed and his mouth half open. He seldom moves his head. He never speaks unless spoken to, but answers questions quite intelligently and distinctly, though slowly and in a low voice. The patient is constantly buttoning and unbuttoning his shirt, or re-arranging the bedclothes, pulling them a little higher, or turning them down for a few inches. His legs and trunk, however, remain quite still. There is no paralysis of facial muscle, of the tongue, or of the limbs. The movements of the eyeballs upwards and downwards and to the right, are performed quite well, but the patient cannot be made to look to the left. Repeated trials fail to induce him to look in that direction; he always states that he is unable to do so, and the eyeballs remain directed straight forwards. (When examined next day this symptom had disappeared and all the movements of the eyeballs could be performed quite well.) Knee-jerks absent, no ankle clonus, plantar reflexes present. No anæsthesia.

Ophthalmoscopic examination revealed distinct optic neuritis in the right eye, but no optic neuritis in the left eye. The condition remained much the same until March 28, when the evening temperature was 101.2° F. On April 2, the morning temperature was 101°, evening 102.8°; April 3, 103.8° in the morning, 104° in the evening. On April 4, the morning temperature was 105.8°, evening 106.4°. The patient became comatose and death occurred on the evening of April 4.

Post-mortem examination revealed a large abscess in the anterior part of the right frontal lobe. It extended to within $\frac{1}{16}$ of an inch of the surface of the anterior extremity of the frontal lobe, and posteriorly to a point a quarter of an inch in front of the right caudate nucleus; laterally it extended to about a quarter of an inch from the convex surface of the cortex, and almost reached the median surface of the frontal lobe. The abscess cavity contained about three ounces of thick creamy pus. The tympanic cavities and petrous portions of each temporal bone were normal. There was no suppuration in either orbit, but a small quantity of pus was found in the upper ethmoidal sinuses of the nose, on the right side. The left sinuses contained no pus. No changes of importance were detected in other parts of the body.

Case 3.—Tumour compressing left frontal lobe; convulsions; mania; coma.—Charles W., age 36, came under my observation when house physician at the Manchester Royal Infirmary in 1884. The patient was brought to the accident room late one evening in December of that year in a comatose state. There

were no unilateral nervous symptoms (such as paresis), no signs of injury to the skull, no peculiar smell of the breath, no indications of disease of the heart or lungs. As no definite history could be obtained, in view of the possibility of some narcotic poisoning, I decided to wash out the stomach. This was done without any improvement following, and the man was afterwards admitted as an in-patient under the care of Dr. Leech. He recovered consciousness gradually next day and was discharged a few days later.

The patient returned to his work and continued fairly well, with the exception of mental dulness, drowsiness and constipation, until June 1, when he had a severe convulsion. He recovered rapidly and after the convulsions ceased, he walked to the Infirmary and was admitted as an in-patient June 1, 1885. From the notes of my friend, Dr. Wild, who was then house physician, I learned that for the first two days after admission he presented no mental symptoms, but on the evening of June 3, symptoms of acute mania developed. He became violent and had delusions of suspicion; later he became more quiet, but remained irritable. On June 4, being allowed to go into the lavatory, he locked the door and jumped through the window. He fell on the grass beneath and immediately got up and commenced to run away. He was seen and captured and brought back to the Infirmary. Beyond a slight bruising of the back, he appeared to have been uninjured. Epileptiform convulsions then commenced and were frequently repeated. In twenty-four hours (from June 5 to 6), he had forty-seven epileptiform convulsions. Between the convulsions he remained comatose. The urine and fæces were passed in the bed involuntarily.

The fits continued for a few days and then gradually ceased, and the patient regained consciousness. He appeared to be in great pain, and frequently made a peculiar wailing noise. Retention of urine came on and a catheter had to be used. In two days after the commencement of the use of the catheter the urine became ammoniacal and contained pus. The patient then improved, the fits ceased, he became conscious and was able to take his food and to pass the catheter himself. The bladder was washed out with a solution of boracic acid daily and the urine became clear and acid. The patient, however, began to emaciate rapidly, and the temperature became raised above normal and irregular. He became much weaker and the retention of urine was succeeded by complete incontinence. He refused food, appeared to suffer from great pain in the head, and often gave a

peculiar wailing cry. The tongue became brown and the teeth covered with sordes. Picking at the bed clothes was noticed, and the patient became completely comatose. Death occurred on July 1.

Autopsy.—Growing from the inner aspect of the dura mater near the anterior portion of the left frontal lobe, was a tumour the size of a large walnut. This tumour had grown into the apex of the left frontal lobe, pushing before it the pia mater which lined the cavity in which the tumour was situated. The brain substance around was not at all infiltrated by the growth, which was circumscribed and could be shelled out easily from the depression in the frontal lobe. On section the tumour was firm and of a reddish grey colour, and contained numerous small spaces (the size of small peas), filled with blood. There was considerable œdema over the surface of the hemisphere, beneath the pia mater. Elsewhere the brain and medulla presented no abnormalities. There was slight tubercular disease at the apex of each lung. Both kidneys were studded with numerous dark red patches, the size of penny pieces. In the liver was a very vascular, dark red, new growth, the size of a large marble. No other changes of importance were found.

The two following cases did not come under my own observation, but are recorded in the clinical reports of the Manchester Royal Infirmary and have never been published.

Case 4.—*Tumour of left frontal lobe, deviation of eye and head to the left; long attacks of profound "sleep."* Ed. B., admitted as an in-patient at the Manchester Royal Infirmary, August 1, 1887, under the care of Dr. Steell.

Previous history.—Five and a-half years previous to admission a boy threw a piece of coal at the patient's head. This struck the occipital region, a scalp wound was produced, but the patient did not lose consciousness. Just when the wound was beginning to heal (five or six weeks after the blow) the patient had a fit. Six months later a second fit occurred and the patient has had five or six fits since. Three months ago he began to be troubled with headache, loss of appetite, sickness and diarrhœa, and his medical attendant ordered him to bed. He slept for two or three days, and it was scarcely possible to rouse him. Similar attacks have occurred once or twice a week since. For several weeks before admission patient is said to have dragged his legs. On July 25 his mental condition became worse, and he was unable to stand. The speech was affected for

about a week before admission. The patient stammered and "made mistakes," "said things the wrong way about." He has complained of pain in the neck for two months.

Present state.—Patient lies with his head and eyes turned to the left. He is very irritable, does not answer questions, appears to be semi-conscious, moans when touched or disturbed. Double optic neuritis is present.

August 4.—Head turned to the left; neck rigid; appears to have much pain in the neck when an attempt is made to turn the head to the right. No paralysis of limbs. Appears to have much pain when the legs are moved; knee-jerks increased.

August 5.—Patient lies with his head still turned to the left. The eyes are half open. When asked "How are you?" he usually answers indistinctly, "I'm all right." There is still much pain when one attempts to rotate the head to the right. He is able to show the teeth and to protrude the tongue quite well. The temperature rose to 104.4° on the evening of August 6; pulse 128, respiration 50. On the morning of August 7 it was 101.2° , in the evening 103.8° . Death occurred on August 8.

Autopsy (head only).—Dura mater normal; pia mater congested, but no signs of lymph or pus at any part thereof. The left frontal lobe was occupied by a new growth which extended to the outer and inner surfaces of the lobe. It projected markedly on the median surface and compressed the median surface of the right frontal lobe. Posteriorly it extended to the anterior part of the left caudate nucleus, but the greater part of that nucleus, together with the lenticular nucleus and internal capsule, was not affected. The growth was very soft and vascular, and the greater part had undergone mucoid degeneration. Other parts of the brain were normal to the naked eye.

Case 5.—*Tumour of right frontal lobe; absence of knee-jerks; occipital headache.* Thos. B.¹ aged 17, admitted June 26, 1888, under the care of Dr. Dreschfeld.

Previous history.—Six years before admission patient began to suffer from epileptic fits. For the first two years he suffered from attacks of *petit mal*. These occurred once or twice a week. Four years ago he began to suffer from attacks of *haut mal*. These attacks occurred sometimes two or three times a week; sometimes he would have four or five attacks daily, and then the attacks would cease for a while. Before each attack patient had an aura, consisting of a "twitching sensation,"

¹ Abstract from notes by Mr. Gore.

running up the left arm from the tips of the finger; this sensation would last five minutes before each attack. Three months before admission the fits ceased. Since that time he has been troubled with pain in the occipital region, which sometimes has passed over to the frontal region. The pain, of a throbbing character, has been constantly present. The vision has become affected considerably; for three months he has suffered from intolerance of light, and on looking at any white object there has been frontal headache. Recently he has been troubled with sleeplessness and loss of memory.

Condition after admission.—The patient lies in a drowsy condition, but is easily roused. The face has a languid expression. The pupils are somewhat dilated, and there is an internal squint of the left eye. He is fairly well nourished. He is intelligent; he answers questions somewhat slowly, but correctly. He suffers from giddiness and constant headache in the inferior occipital region. When he looks at anything white he suffers from frontal headache. The pain in the occipital region is of a throbbing character. There is slight paralysis of the lower facial muscles of the left side, and the left orbicularis palpebrarum is feebler than the right. The movements of the left arm and leg are somewhat weaker than those of the right. The tongue is protruded straight. The epigastric, abdominal cremasteric reflexes present on both sides, better marked on the right side than on the left. Plantar reflexes increased. Knee-jerks *absent*. There is no anæsthesia. The gait is staggering; the patient tends to fall towards the right side, and has a sensation of vertigo. He cannot stand upright with his eyes closed. Nothing of importance detected on physical examination of the heart and abdomen. Urine: no albumen, no sugar. Slight dulness at apex of left lung.

June 29.—Marked double optic neuritis.

July 4.—Patient was seized with an attack of rigidity of the muscles of the body, the head was thrown back, the arms, legs, and back were rigid. The attack lasted for about one minute; the patient never lost consciousness completely.

July 13.—Patient has complained of severe headache during the last two days; he complains of pain shooting from the occipital to the frontal region. He is in a drowsy condition most of the day.

July 30.—Patient is very drowsy, almost comatose. He answers questions slowly. Complains of much pain on pressure over the back of the neck. Muscles of the neck rigid; head retracted. Death on August 6.

Autopsy.—Cicatrix of healed phthisis at apex of right lung. A small tubercular cavity at apex of left lung, and several small patches of consolidation around.

Brain.—In the right frontal lobe was a roughly spherical tumour two and a half inches in diameter, which came to the surface in the region of the first and second frontal convolutions; other parts of the brain not affected. Cerebellum normal. No growth at the base of the brain. The growth was *firm*, but was not hard except at the centre. The periphery was of a pale greyish red colour and vascular; the centre was pale and somewhat caseous in appearance; at various parts were masses of calcareous material. The growth could be easily shelled out from the surrounding softened brain substance.

The vertebræ, spinal dura mater, and spinal cord were all carefully examined with negative results. No changes of importance in the other organs.

REMARKS.—In two of the cases reported it is interesting to note that optic neuritis was unilateral; in one case on the side of the lesion, in the other it was unilateral though *both* frontal lobes were invaded by tumour growth. Another point of interest is the absence of knee-jerks in three of the five cases above reported. In one of these cases the spinal cord was examined microscopically, and was found to be normal, and it is probable, from the clinical history, that in the other cases the cord would have been found normal had a microscopical examination been made.

The severe pain in the occipital region in Case 5 was somewhat peculiar. In Case 4 the deviation of the head and eyes towards the side of the lesion was a symptom of interest, since Ferrier and others localise the centre for this movement to the præ-frontal region. The very long attacks of "deep sleep" are also of interest in this case.

All these points will be referred to subsequently, since the symptoms of the five cases above recorded are included in the following analysis.

ANALYSIS OF THE SYMPTOMS, &c., IN 50 CASES OF TUMOUR OR ABSCESS INVOLVING THE PRÆ-FRONTAL LOBES.

The cases comprise the 5 reported above, and 45 recorded in medical literature in recent years.¹

Nature.—Of these 50 cases 4 were cases of abscess, and the rest tumours of various kinds—10 of the latter were gliomatous,

¹ For references see list at the end of the paper.

7 sarcomatous, 5 gliosarcomatous, 4 syphilitic, 3 endotheliomatous, 2 tubercular.

Region.—17 involved the right lobe, 22 the left, and 11 both lobes. The convex cortex was the seat of the growth in 18 cases, the median cortex in 1, the inferior cortex in 2, the white matter in 8, the convexity and the white matter in 5. In 3 the growth was at the tip of the anterior part of the frontal lobe; in 3 it was between the lobes; and in 10 it was situated at the base and compressed the frontal lobe.

Symptoms.

Headache was a prominent symptom in most cases. The pain was generally most intense in the frontal region, in some cases in the occipital region. In the records of 50 cases headache is not mentioned, or the locality not stated, in 23; in 1 there was no headache; in 1 the region of the pain varied. In the remaining 25 cases the distribution of the headache was as follows:—

	Cases.
Frontal region only, in	11
* { Frontal chiefly, occipital also	4
{ Occipital chiefly, frontal also	2
{ Occipital at first, frontal later	1
{ Occipital	2
Frontal and vertical	2
Vertical and temporal (near the growth)	1
Temporal (growth on opposite side)	1
Headache "on the side of the tumour"	1

* In 9 of the 25 cases pain was in the occipital region, with or without frontal headache = 36 per cent. of cases in which headache was *localised*.

Frontal headache is therefore most frequent, but it is remarkable that the occipital headache also should often occur. It is well known that in cerebellar tumour the headache is often occipital, and not infrequently it is frontal or frontal and occipital. Dr. Purves Stewart (*Edinburgh Medical Journal*, January, 1896, p. 659), has called attention to unilateral frontal headache in cerebellar tumour situated in one lateral lobe, the headache in the frontal region being on the side opposite to the cerebellar lesion.

Tenderness on Percussion of the Skull.—In 30 out of the 50 cases there is no note with regard to this symptom. In the other 20 cases it is stated, that there was tenderness on percussion of the skull in the *frontal* region in 15 cases. In one of the cases the occiput was tender also, but the tenderness was most marked in the frontal region. In 5 cases it is stated that there

was no tenderness of the skull on percussion. Of those 15 cases in which there was tenderness on percussion of the skull, the tumour growth was situated at or near the region of the tender area in 12. In 1 there was tenderness on one side, and both frontal lobes were the seat of tumour growth.

Hence it appears that if tenderness be present on percussion, it is of value as an indication of the region of the skull in which the growth is situated. Also when tenderness is present in the frontal region, if it be unilateral, the growth is generally on that *side*.

Tenderness on percussion is not a sure indication that the growth is cortical, though this is generally the case. Thus in the 15 cases in which there was tenderness on percussion the growth was superficial (cortical or in the meninges) in 12, but in 3 it was in the white matter. Again, in the 5 cases in which there was no tenderness the growth was cortical in 3, seated in the white matter in 2.

Ophthalmoscopic Examination.—In the 50 cases analysed, the records of ophthalmoscopic examination were as follows:—

	Cases.
Double optic neuritis, in	25
One eye previously lost; optic neuritis in the remaining eye, in	1
Vision impaired in both eyes, in	1
Vision lost in both eyes, in	1
Condition of optic discs and vision not mentioned, in ..	11
Optic discs normal, in	4
Optic neuritis on one side only (the side of the lesion), in ..	1
Optic neuritis unilateral, lesion in <i>both</i> frontal lobes, in ..	1
Optic neuritis very much greater on side of the lesion, in ..	2
* Blindness on the side of the lesion; the other eye only affected	
at a later date, in	1
Well marked primary optic atrophy on the side of the lesion;	
slight optic neuritis on the other side, in	2

* Hence in 7 out of the 35 cases in which ocular changes were noted, the failure of sight or the ophthalmoscopic changes were on one side only, or were very much more marked on one side. In 6 of these 7 cases, the marked ocular changes were on *the side* of the lesion; in one case there was unilateral optic neuritis and a bilateral lesion.

This marked unilateral tendency, or marked difference on the two sides, in 7 out of 35 cases is of some interest, since such a difference does not occur, or does not occur in this proportion of cases, in lesions of other parts of the brain substance.

Sense of Smell.—In 33 out of the 50 cases no note with respect to the sense of smell is given. In 10 cases it is stated that smell was not affected. In 7 there was unilateral or bilateral loss of smell, and in these cases the growth involved the basal region, *i.e.*, region of the olfactory nerves.

Loss of smell, if present, is therefore an important symptom, but if the sense of smell be normal no conclusion can be drawn from this fact alone.

Exophthalmos.—This symptom was bilateral in 1 case, unilateral (on the sides of the growth) in 2 other cases. In all these cases the growth had invaded the dura mater and anterior fossa of the skull.

Localised Swelling in the frontal region of the skull was recorded in one case; in another case there was an external swelling in the temporal region, at the lower angle of the frontal lobe.

Motor Symptoms.—Paresis, or paralysis of the face or arm or leg, or of all three parts, on the side opposite to the lesion, was met with in many cases at some period; but in the majority it was very slight, and was often noticed *only* at the end of the illness. The paresis was due to the extension of the growth backwards from the præ-frontal towards the motor area of the cortex, or to softening around the tumour growth implicating the motor area or the motor fibres coming from that region.

	Cases.
Slight paresis on side opposite to the lesion, noted at some period, in	27
Marked paralysis, in	4
Motor symptoms absent, in	5
Motor symptoms not mentioned, in	14

Convulsions were met with in many cases; their nature is shown in the following table. The frequency of the attacks varied greatly.

	Cases.
General convulsions occurred at some time in the course of the patient's illness, in	14
Unilateral convulsions from involvement of the motor area of the cortex, in	10
* Convulsive attacks (clonic spasms) in both arms, in	3
Fits resembling those of hystero-epilepsy, in	1
Fits beginning by the legs being drawn up into the lithotomy position, in	1
Attacks of tonic spasms in the neck	2
Convulsions absent, in	4
Convulsions not mentioned, in	15
	50
	—

*In two of these cases the growth was median and involved the median surface of both frontal lobes. In one case there was a lesion in the præ-frontal area, near the arm centre, in *each* hemisphere.

Anæsthesia was never recorded; in many cases it is definitely stated that sensation was not affected, in others no note is made.

Ataxia.—Bruns ⁽²⁰⁾ has drawn attention to the fact that ataxia resembling that of cerebellar disease is sometimes met with in lesions of the præ-frontal area.

In the 50 cases analysed, ataxia, unsteadiness in walking, or a reeling gait was present in 14; these symptoms were not mentioned in 33, and in 3 cases it was stated that no ataxia was present.

Knee-jerks.—It has been long known that the knee-jerks are sometimes absent in cerebellar lesions, whilst in other cases they are increased; why this difference should occur is not known. It is interesting to note that the knee-jerks are sometimes absent in frontal tumour also; in the 50 cases the condition was as follows:—

	Cases.
Knee-jerks absent on both sides, in	6
Knee-jerks very feeble on both sides, in	2
Knee-jerk very feeble on side opposite to tumour, in	4
Knee-jerk very feeble on side of tumour, in	2
Knee-jerks normal or increased, in	16
Knee-jerks not mentioned, in	20

In 30 cases only was the condition of the knee-jerks referred to, and the proportion of cases in which they were absent, amongst these 30 cases was 20 per cent. (6 in 30).

In all the cases in which the knee-jerks were absent, there was no indication of the lesion of the spinal cord or peripheral nerves, but in only one of the cases, the first reported in this paper, was the absence of microscopical changes verified by pathological examination. The absence of knee-jerks is a symptom of interest. In tumour growths involving the motor region of the brain, the knee-jerk on the opposite side is generally increased, and on the side of the lesion, present, if not increased. In 30 of such cases taken for comparison with the 30 lesions of the præ-frontal area analysed above, I found the knee-jerks both present in all except one, and in this case the growth was very extensive and invaded the basal ganglia. The connection of each lateral cerebellar lobe with the præ-frontal region of the opposite side,

is of interest with respect to the loss of long knee-jerks that sometimes occurs in lesion of both these regions.

Mental Symptoms.—The psychical symptoms which have been observed after removal of the frontal lobes in animals are referred to at the commencement of this paper, and it is interesting to note, that clinically mental symptoms have been noted in most cases of lesions of the frontal lobes, though the exact nature of these symptoms has varied somewhat. A dull mental condition, mental decadence, loss of attention, a drowsy, and semi-comatose state have often been recorded.

Some writers have attached considerable importance to a childish behaviour of the patient, with an abnormal tendency to fall asleep.

Jastrowitz²³ has described a peculiar mental condition which he has met with in cases of lesion of the præ-frontal lobes, a condition of mental impairment or dementia along with a peculiar cheerfulness or hilarity, and tendency to jest and make puns (*Verhandlungen des Vereins f. innere Medicin*, Berlin, 1888). He acknowledges that this mental condition is not met with in all cases, and that it occurs also in other diseases, such as general paralysis of the insane and in chronic alcoholism; but he thinks that in a case presenting general symptoms of intra-cranial growth, the tumour may be localised in the frontal lobe, if this peculiar mental condition should be present.

Oppenheim²³ also draws attention to this peculiar humorous mental condition in cases of frontal tumour, and points out that the cheerful mental state stands out in sharp contrast to the miserable physical condition of the patient. There is a tendency to joke, to make sarcastic or trivial answers; Oppenheim compares the mental condition to that of a patient awaking imperfectly from a pleasant narcosis. He adds that it must be allowed that this mental condition occurs principally in tumours of the frontal lobes.

Bruns²⁰ also describes a similar mental state.

Welt (*Deutsches Archiv. f. Klin. Med.*, Bd. 42, 1888, p. 339) attaches importance to an irritable mental condition, with violent and mischievous behaviour, especially in lesions of the convolutions on the orbital surface.

Lloyd (*Journal of Nervous and Mental Diseases*, p. 81) draws attention to a peculiar "inhibition of thought; an increase of the time reaction of cerebration;" a peculiar slowness in answering questions, though the answers are quite intelligent.

In the fifty cases which I have analysed, mental symptoms were generally well marked; in many cases they were the earliest and most prominent symptoms. The following table shows the varieties of mental condition met with:—

	Cases.
* A condition of mental decadence; a dull mental state; loss of power of attention; loss of memory; loss of spontaneity; the patient taking no notice of his surroundings; sleeping during the greater portion of the day, or being semi-comatose	32
Loss memory, mental failure, but patient cheerful	6
Patient suspicious; suffered from delusion, and was occasionally violent	1
Patient irritable and violent	1
Patient generally asleep; irritable when awake	2
Patient ambitious, excitable, memory lost	1
Slowness of mental processes; patient simple and childish	1
Mental anxiety; childishness; hallucinations; suicidal tendencies	1
Mental condition not stated	5
	<hr/> 50 <hr/>

* In two of these it is noted that the patients were in a perplexed mental condition, and constantly appeared to be searching for something.

In some cases in which the mental symptoms were well marked, the lesion was chiefly in the white matter, in some cases, chiefly in the cortex, whilst in others both cortex and white matter were involved; so that one does not feel justified in drawing any conclusion as to whether the mental symptoms are the result of lesion of the cortex or of the white matter. In 3 cases in which the lesion was chiefly cortical, the mental symptoms were comparatively slight.

Loss of power in the *muscles of the back* is not mentioned in any of the records.

Sudden onset of Symptoms.—Sudden coma, when only slight and trivial symptoms had been complained of previously, was recorded in 3 cases.

Attacks of Coma.—As a rule the patient finally became

comatose, and in many cases was comatose or semi-comatose for a long period before death. But apart from this terminal comatose state, in some cases the patient suffered from one or more previous attacks of coma, without any preceding convulsions. Sometimes these attacks were followed by hemi-paresis; in other cases no paresis followed. Attacks of unconsciousness previous to the final coma were noted in 10 out of the 50 cases analysed.

Remarks on Diagnosis.—In a case in which there are general symptoms of intra-cranial growth or abscess, the localisation to the præ-frontal region often presents considerable difficulty. The diagnosis is sometimes especially difficult between cerebellar and frontal tumours. In tumours of the præ-frontal region, whilst the headache is chiefly frontal, very often there is occipital headache also. In cerebellar tumours whilst the headache is chiefly occipital, still not infrequently there is frontal headache. In tumours of the cerebellum the knee-jerks are sometimes absent, but as shown by the cases recorded in this paper, the knee-jerks are sometimes absent in lesions of the præ-frontal region. Also ataxia, simulating the ataxia of cerebellar disease, may occur in lesions of the præ-frontal region. Each præ-frontal lobe of the cerebrum is connected with the lateral lobe of the cerebellum of the opposite side, by fibres passing downwards in the anterior part of the internal capsule, on the inner side of the crus cerebri, and in the superior cerebellar peduncle; and this connection may, in some way, account for the above-mentioned symptoms being often common to lesion of both the præ-frontal region and the cerebellum.

In many cases, however, a careful study of the symptoms will lead to a correct localisation. Mental symptoms (especially mental dulness and degeneration) are prominent, and often early symptoms in lesions of the præ-frontal regions, whilst they are generally slight and occur late in lesions of the cerebellum. Paresis on one side—hemi-paresis, or paresis of leg, arm or face only—occurs in a large number of cases of præ-frontal lesions at a late period, owing to the extension backward of the growth, or of the surrounding

softening; whilst paresis of the arms or legs is very rare in cerebellar lesion. Convulsions, general or localised, often occur at some period in præ-frontal lesion; very rarely in cerebellar lesion.

Tenderness on percussion or pressure over the frontal region appears to be a sign of value in localising the lesion to the præ-frontal region.

Whilst optic neuritis is often bilateral in præ-frontal tumour and abscesses, still the facts above recorded show that in some cases it is unilateral, or it is *very much* more advanced on one side, or vision is *very much* more impaired on one side, or there is primary optic atrophy on one side, and only slight optic neuritis on the other. I have never met with any case of cerebellar tumour in which there was this tendency to very marked affection of one optic disc, whilst the other was normal, or only very slightly affected.

[It is interesting to note that the lesion has been on the side on which the optic disc was markedly affected in 6 out of 7 of these cases; in the seventh the lesion was bilateral]. When present, this marked difference of the two optic discs appears to be of some diagnostic importance.

Localised swelling in the frontal region, unilateral exophthalmus and loss of smell are rare symptoms, but, if present, form important evidence in favour of a lesion of the præ-frontal region.

It is to be noted, however, that loss of smell has been reported in cerebellar tumour, but of course is exceedingly rare, whilst it is reported in 7 out of 17 cases of præ-frontal tumours.

With regard to the diagnosis between lesions of the præ-frontal region and the so-called motor area, evidence in favour of localisation in the former would be the prominence and early onset of mental symptoms; the absence or slight nature of motor symptoms, and the development of these symptoms, when present, at a late period, after the general and mental symptoms have become well-marked. Whereas in lesions of the motor area, mental symptoms are often absent or slight until a late period; paresis is an early symptom and develops into well-marked paralysis. Also the

tenderness or percussion over the frontal region, and (in a few cases) the absence of knee-jerks, the loss of smell, the unilateral nature of the ophthalmoscopic changes, or very marked difference of these changes on the two sides, would be points in favour of a lesion of the præ-frontal region.

Suitability for Operative Treatment.—In the 50 cases analysed, the 4 cases of abscess were all suitable cases for surgical interference. One case,³² McEwen's, was operated on with success. Seven cases of tumour of the præ-frontal lobes were so suitable and of such a moderate size, *even at the time of the autopsy*, that they could have been easily removed by surgical operation. In 2 other cases, a tumour was removed; in one case successfully (Durante), in the other unsuccessfully.

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A CONTRIBUTION TO THE PATHOLOGY OF
EPILEPSY. A REPORT OF TWO CASES IN
WHICH A PORTION OF THE BRAIN CORTEX
WAS EXCISED AND EXAMINED.¹

*The Microscopical Examination of the first Case by
Dr. A. Wiener.*

BY JOSEPH COLLINS, M.D., OF NEW YORK.

Neurologist to the City Hospital; Physician to St. Mark's Hospital.

A CAREFUL study of a large part of all that has been written on the value of trephining for epilepsy has convinced me that it is quite impossible to gather from the literature alone what the value of operative interference in epilepsy may be. This is so for many reasons. In the first place, the significance given to the designation "operation for epilepsy" is a variable one. Leaving aside the operations that are done on other parts of the body such as the eyes, the nose, &c., for the cure of epilepsy by removing so-called reflex sources of the disease and confining our criticisms to operations on the skull, we note that for one writer operation in epilepsy means simple trephining; for another, trephining and removal of the dura; while for a third, it means not only both of these procedures, but in addition, the excision of a portion of the cortex, whether or not it be the seat of apparent disease. When such radically different interpretations are put upon the words "operation for epilepsy," it will be seen how extremely difficult it is to gather any reliable data as to the value of what we consider operative procedure in epilepsy; that is the value of making a large opening in the skull, of dissecting the dura, and of removing

¹ Read before the American Neurological Association, June, 1896.

the cortical area which has been shown by clinical observation and experimental test to be the seat of the lesion.

At the present day there would seem to be a tendency to absolutely decry the value of all forms of operative procedure in the treatment of epilepsy. This attitude of the profession is the natural reaction to the intemperate espousal of the supposed usefulness of operation which occurred a few years ago. Unfortunately, during this over-enthusiastic period in the early development of brain surgery, many cases of epilepsy were operated on, in one of the three ways mentioned above, in which there was no indication for operation, and in which no hypothesis could be formulated to explain why such an operation could be of benefit. These cases have contributed to establish the verdict of "no utility."

Arrays of statistics have been gathered by different writers to prove or to disprove the value of operation on the skull in epilepsy; but in almost every instance there is such utter lack of discrimination in selecting the cases that should be included, or excluded, that such tabulations are worse than useless; they are positively baneful, for by manipulation they may be made to bear evidence for or against the value of operation. In addition to this charge, the hackneyed one of premature report of cases that have been operated on must be mentioned. It is so ridiculous for a man with scientific training to report the favourable or unfavourable issue of a case in which operation on the head for the relief of a disease like epilepsy has been done, a few weeks after the operation, that one is chagrined even to have to refer to these cases. Yet a large number of such cases have been, and are reported, and statisticians to whom the critical faculty is as foreign as is the nature of evidence, use such cases to settle the question of value of trephining operations!

I would not be understood as saying that there are no cases on record in which indications for operation in the sense in which we use the term, viz., making a large episcranial bone flap, dissecting off the dura and excising a piece of the cortex, have not been clearly defined, and in which the operation has been masterfully done. There are

such cases, but they comprise the great minority in the literature on the subject.

The writer believes that the indications for operation in epilepsy are very limited, but that such indications occur not only in Jacksonian epilepsy, but in so-called idiopathic epilepsy.¹ Operation is indicated in Jacksonian epilepsy when there are evidences of irritation of a circumscribed motor cortical area, and it matters not what such irritation may be caused by. The source of irritation, as well as the surrounding cortex for a considerable distance, should be extirpated, and the sooner such extirpation follows the first attack of localised spasm, the greater will be the chances of absolute cure.

Operation is indicated in idiopathic focal epilepsy when the convulsive attack begins always with the same localised spasm, and especially if, in the early stages of the disease, such localised spasm be the only convulsive manifestation.

It is the purpose of this paper to elicit your criticism of two cases, the first conforming to the type of Jacksonian epilepsy, and the second to that of idiopathic epilepsy.

The points to which I wish particularly to direct attention in the first case are: the careful clinical study, the exact location of the lesion, the early operation—for the patient had no more than three attacks altogether, and only one of them passed into a typical epileptic convulsion—and, lastly, to the pathological changes which were found in the brain, and the meninges and the causal relationship existing between them and the attacks of epilepsy. The transference of the recently removed cortical matter directly into absolute alcohol, thus avoiding to as great extent as possible the formation of artificial changes, and the use of the most approved technic for the detection of changes in the cortex, were important factors in contributing a satisfactory explanation of the case. The probability of complete cessation of

¹ The use of the term, idiopathic epilepsy, for these cases is deprecated, for idiopathic epilepsy is used synonymously with inherited epilepsy by most writers. If these cases could be called focal epilepsy, inasmuch as the motor or sensory discharge of energy points to a localised area in the brain, without confounding them with Jacksonian epilepsy, which is always focal, and in which some lesion, apart from the supposed change in the large cells of the pyramids, exists, it would be very desirable.

the symptoms of epilepsy, now that the diseased area is removed, will also be touched on.

CASE I.

History.—O. L., 20 years old, an American of German parentage, consulted me April 19, 1895. The family history showed on the maternal side mental trouble. The patient had one younger brother defective mentally, who died of spinal disease. The patient is the fourth child of the family. During infancy he was free from convulsions and serious disease. He went to school until his fifteenth year, and was considered moderately bright. He had scarlet fever and diphtheria six years ago and made a good recovery from each of them. He was well till November 29, 1894. At that time he was working in a theatre selling librettos. On the evening of that day while sitting home reading a newspaper, he felt a sensation arising in the fingers of the right hand like an electric shock, associated with a twitching of the thumb and index finger. This lasted about three minutes. The sensation went up to the side of his face, then he lost consciousness. He had in short, a typical epileptiform attack. There was no frothing at the mouth, but he bit the tongue. After the attack he was prostrated and wanted to sleep. He felt so bad that he did not go to the theatre that night. His attack was looked upon as nervousness and fright but, nevertheless, his parents kept him home for several days. Two or three times that week he had some twitchings of the fingers, less severe than the first and not accompanied by loss of consciousness. He had no further trouble or complaint until two or three weeks before coming to me, that is in March 1895, when he experienced a numb feeling in the right hand, this would come and go quickly. He says the sensation was like a shock; all the strength, the natural feeling seemed to go out of the hand. Three days ago, while walking in the street he felt twitchings in the right hand, accompanied by the same numb feeling which he had before experienced. The twitching lasted ten minutes and was confined principally to the index finger and thumb. A premonitory pricking sensation of a few seconds

duration seemed to herald it. Then after a short time a second attack of the twitching occurred. He did not lose consciousness but felt bad after the attack.

There is no history of recent injury to head of any import. Some four or five years ago while practising in a gymnasium he was thrown against the wall, and although stunned for a few minutes there was no loss of consciousness nor was there headache following it.

Examination.—No defect of station or gait. Both knee jerks lively, right exaggerated. Considerable ankle clonus on right side, absent on left. Grip of right hand weaker than left and weaker than formerly. Triceps reflex on right side livelier than on left. Myotatic irritability of entire right half of body greater than left. Does not complain of cephalic paresthesiæ or headache. There are no scars on the surface of the head. No tenderness on percussion of scalp. Has some marks of degeneracy such as vaulted palatine arch, non-lobulated ears, clubbed finger-ends, and coarse hair. There is slight tremor of the right hand and a little uncertainty of movement, not sufficient, however, to be called ataxia. Sensibility of right hand and right upper extremity, in fact, of every part of the body seems to be intact. The patient was referred to Dr. Knapp, for examination of the eyes, who reported as follows :—

The patient, Mr. L. whom you sent to me for examination, shows the following conditions :—

V. $\frac{20}{40}$ brought up to $\frac{20}{20}$ by glass correcting a low degree of myopia and myopic astigmatism (R. 0.50 sph., L. 1.00, cy. 1.80; muscles out of balance, patient showing a small amount of esophoria both for distance and near.

Muscular and refractive condition unaffected by complete relaxation of the accommodation with homatropine. Field for white and for all colours normal in each eye. Fundus normal in ophthalmoscopic examination both with and without a mydriatic.

A diagnosis of Jacksonian epilepsy was made, and on account of the absence of the cardinal symptoms of brain tumour, such as optic neuritis, headache, vomiting, etc., the pathological diagnosis was meningo-encephalitis. Operation

was urgently advised. As it did not seem to the parents that the boy was ill there was considerable hesitancy and delay on their part in consenting to such a procedure.

In the meantime the patient was taken to Dr. B. Sachs, who made the same diagnosis and recommended the same measure for relief. The patient was admitted to Mount Sinai Hospital on May 9, Dr. Gerster having consented to perform the operation. The patient was prepared in the usual manner, and the topography of the right hand area particularly the thumb and finger area was outlined on the left side of the skull. Dr. Gerster proposed to do the bone flap operation. The skull was found to be extremely thick, and the procedure of cutting through it with the mallet and chisel was a very slow one. The patient did not seem to tolerate the ether very well, and frequently it was necessary toward the end of the operation to cease work as it was noticed that after every few blows of the mallet the pulse beat would diminish, in some instances rather alarmingly. It was found impracticable to make a bone flap so the section of bone was removed. The dura was not adherent, and it with its vessels, presented a very normal appearance. The cortex was irritated by a weak faradic current and flexion movements of the hand, exquisitely in the index finger and thumb were produced. On account of the time consumed in getting into the brain cavity and the condition of the patient, it was thought wise to postpone further investigation and operation for a few days. The wound was dressed with a simple aseptic dressing. The patient rallied well, aided by moderate stimulation, and the next day he had an evening temperature of 101.6. On the second day after this preliminary operation he had an attack beginning with twitching in the face and right hand and forearm; this latter lasted for about ten minutes, when he became subject to a rather violent general epileptic seizure, the convulsive part of which lasted about ten minutes. He did not, however, regain consciousness, and after a quarter-of-an-hour he had a second severe convulsive seizure, after which he slowly recovered consciousness. After the preliminary operation the right hand seemed to be quite powerless, although no

part of the cortex had been removed. Sensibility of the right hand remained as before. Four days after the first operation, May 17, the second operation was done. On removal of the bandages it was seen that there was marked œdema of the pericranial tissues extending over the entire right surface of the skull. The wound itself was healthy in appearance. A flap was raised from the dura over an area corresponding to the original opening and it seemed to be quite normal. That part of the central convolutions which on being irritated with the faradic current caused a contraction of the fingers of the right hand was then excised. Two pieces of cortical tissue each about the size of the terminal phalanx of the thumb were removed.

The patient tolerated the second operation very much better than the first, and at neither was any considerable amount of blood lost. The wound was closed by simply apposing the epicranial flaps and covered with the customary surgical dressings. On the day following the second operation the patient felt quite well. Examination showed well-marked paralysis of the right hand, upper extremity and paresis of the right side of the face. Pain sensations could be quite well located in the right hand, contact sensations to a much lesser degree. On the fourth day after the operation he had a mild attack beginning with twitching movements around the right eye and right side of the face, and then becoming general. Later in the day he had a severe general attack in which he was unconscious for about an hour. After recovery from this there was an attack lasting about three minutes, in which the twitching was limited to the right side of the face, nose and eyelid. After this attack the motor paralytic symptoms began to mitigate and within a week they had become comparatively slight. Coincident with the disappearance of the motor paralysis, a paralysis of sensation seemed to develop in the right index finger and thumb, and a very considerable ataxia in the entire hand.

From a surgical standpoint the patient made excellent progress, and was discharged from the hospital June 12th. At no time after the second operation did the temperature

go above 100° F. except on the ninth and tenth day, when the thermometer registered a fraction above that figure.

Examination, July 9, 1895.—The patient is active and bright mentally, and strong physically. He does not make any complaint except of a feeling of weight in the right hand. Gait and station are normal. Dynamometer, right hand 70. Left hand 90. The knee jerks are both lively, the right slow in action but it moves through a large arc. There is no ankle clonus, the right hand and fingers show some ataxia and there is a slight perversion of tactile sense, loss of dexterity in grasping anything such as a knife or in picking up a number of coins in succession, but slight diminution of the sense of position. Muscular sense is considerably diminished in the right upper extremity.

July 21. After full warm bath had a *petit mal* attack. Right hand became numb, and the hand drew together with flexed fingers. Speech became thick and inarticulate. One half of tongue, the right, felt numb, and was the seat of a severe pricking sensation. The attack lasted two or three minutes, but there was no dizziness or loss of consciousness, nor did he sleep or become stuporous afterward. Three or four days later he went to see Dr. Gerster who found a strip of gauze in the wound which had prevented it from completely uniting. Since the last visit he has gained four pounds in weight, memory is good, and he is apparently in good shape intellectually.

Dynamometer, right hand 65, left 85. Dexterity, grace, and manipulatory skill in right hand much less than in the left. Has noticed that since the last attack he cannot compress the middle finger of right hand; all the other fingers can be forcibly flexed so that he has a good grip except the middle finger. Tactile sense is lost in this hand. Sense of contact seems to be preserved, that is when a coin is dropped in the right hand he knows when it touches the hand, but he cannot tell when it is removed. Sense of position and muscular sense are likewise injured only in a slight degree. Ataxia of fingers in right hand marked, for instance when told to bring the end of each finger successively in contact with the end of thumb, he gets all mixed. Likewise when

he essays to touch the tip of nose. Can feel cotton all over hands. He is taking now thirty to forty drops of a saturated solution of potassium bromide three times a day.

September 2, 1895.—The right hand is becoming less adroit, especially the fingers. The ataxia and certainty of purposive movements of fingers is somewhat greater. He is now taking sixty drops of a saturated solution of bromide at bedtime. This quantity is reduced to fifty. There have been no attacks. He has no sensations of vertigo, mental confusion, or fainting spells. He feels strong and well, and is at work. Memory and other intellectual faculties seem normal. After the operation he noticed that the toe of the right shoe was worn more than left; this is no longer so. He can use the right hand moderately well. Has power in the whole hand, he says, but in the individual fingers it fails him. Dynamometer, right 20, left 90. There is loss of sense position in the right hand and fingers; but this is not complete. When all the fingers are flexed he cannot extend the fingers individually, nor in pairs, they must all be extended together. Extensor force is less than flexor in fingers, and in wrist. Articular sense in right hand is fairly good. The fingers of the right hand have a tendency to remain flexed with closed fist. When he squeezes anything with left hand, the fingers of right flex into palm (showing motorial impulse from left side goes down right). Tactile sense all right; can tell the moment anything touches his hand. Has good sense of weight and temperature. Knee jerks right side slightly exaggerated. Tongue deviates slightly to left. Face not so completely and thoroughly innervated on right side as on left. Triceps reflex right side exaggerated, and the myotatic irritability of the right upper extremity is increased. The strength of the lower extremities seems to be equal on both sides. He has been taking twenty-five grains of bromide daily, one half the quantity in the early evening, the other half on retiring.

Examination on June 1 of the present year, somewhat more than a year after the operation, and very nearly a year after the date of the last epileptic attack, shows that the

strength of the right hand causes the dynamometer to register 30, and with the left hand 100. Myotatic irritability of the right upper extremity is increased. There is slight exaggeration of the right knee-jerk, but there is no ankle or patellar clonus. There is no ataxia of gait; the patient stands well with the feet together, and on either foot alone. Examination of sensibility shows that the sense of touch in the right upper extremity is exquisitely preserved, likewise the sense of pain and temperature. Muscular sense in the hand is somewhat diminished, and the sense of position is not perfect; but aside from these there are no sensory shortcomings. The right hand has a tendency to remain in a semi-flexed position, and the fingers cannot be extended individually, but they can collectively. There is very little ataxia of the right hand.

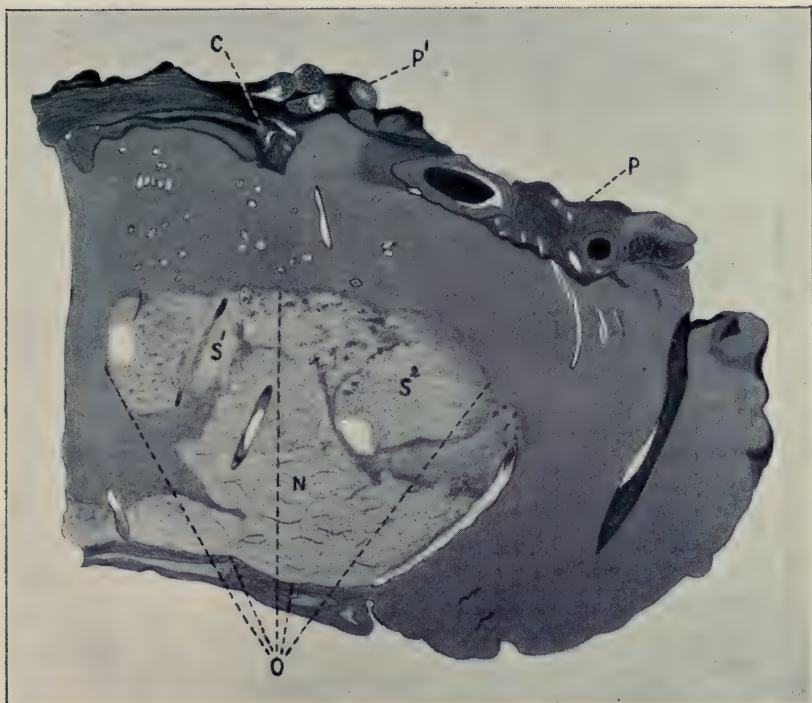
The patient is engaged in clerical work; he has learned to write with the left hand, and, aside from the inconvenience attending from a loss of dexterity with the right hand, he may be said to be in perfect health. During the past four months the patient has been taking twenty grains of bromide of potassium once a day, on retiring; this, with the ordinary measures for the maintenance of a commendable nutrition is all that is being done for him. There have been no attacks of vertigo, no recurring sensations of numbness and loss of strength in the hand, no attacks of mental confusion, nor, in short, anything that could be construed as a motor, or psycho-motor discharge of energy resembling an epileptiform attack. There has been no mental deterioration whatever; in fact, it is probable that the mental faculties of the patient are more active now than they were before the operation.

It may be said, and if so, no effort will be made to controvert it, that the quantity of bromide of potassium which the patient has taken has been sufficient to hold the epileptic attacks in abeyance. If the patient remains in my care it is proposed to keep up the quantity of bromide which he is now taking for at least two years longer, that is, providing that within that time no epileptiform manifestation shows itself. If it should, the dose of bromide will be materially increased. In the opinion of the writer, it would be most

unjustifiable not to continue the use of bromide for a long period after the operation in a case of this kind, even when it is believed that the focal origin of the disease has been entirely removed, as it probably was in this instance. It is thought that by such medicinal measures the motor cells in the neighbouring cortex, which were participants in the few convulsive seizures from which the patient suffered, can be so steadied as to prevent further tendencies to explosion which they would naturally have on account of such explosion having previously taken place.

The cortical mass removed in this case measured about four ccm. in length and one and one half ccm. in thickness. The mass was divided into pieces measuring one half ccm. in thickness, and then placed in absolute alcohol. The Nissl stain was used for study of the cells, while for the tissue proper the hæmatoxylineosin and the borax carmin methods were used.

A general view of the specimen taken from the most striking part of the lesion, shows marked pathological change in the pia, in the blood vessels, in the subpial space, and in the cortex, (see fig. 1). The pia is thickened and intimately attached to the cortex; immediately under the pia there appears a diseased cortical area; directly beneath this diseased cortical area there is a fairly normal portion, and then just at the junction of the grey and white matter, there is a distinct area of softening combined with increased neuroglia measuring about one half ccm. in length, and one quarter ccm. in breadth. The pia on closer microscopical examination is seen to be very much thickened; there is considerable formation of connective tissue. The distended blood vessels show thickened walls infiltrated with spheroidal cells. Some of the blood vessels are entirely occluded. Throughout the whole region the pia is closely attached to the cortex; in some places there is an actual growth of connective tissue from the pia into the cortex, completely disorganising it; in other places the pia appears to be merely glued to the surface of the cortex by a large number of spheroidal cells, which shows that in this region a more or less acute process had occurred. This might be accounted

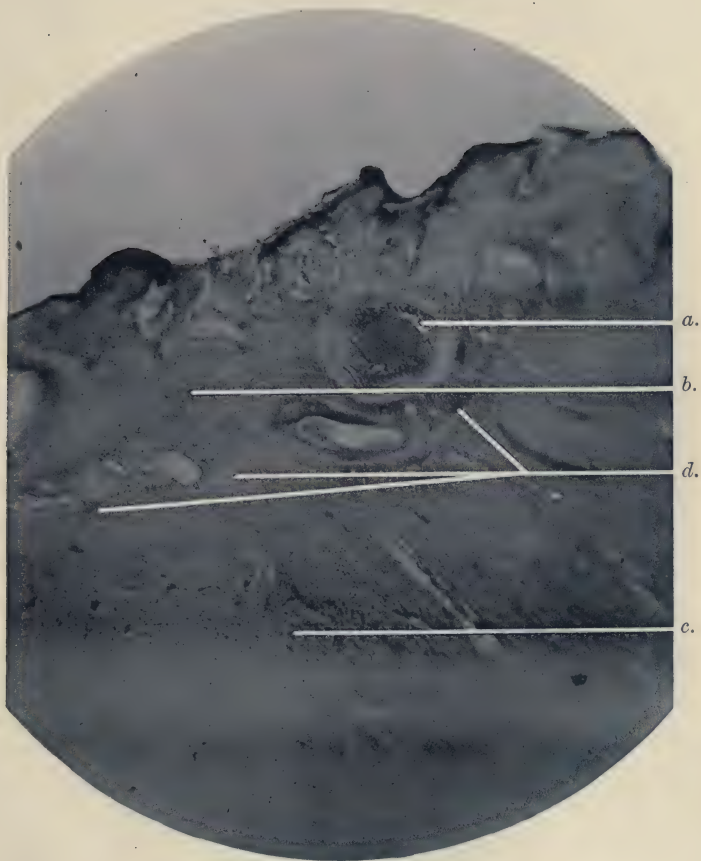


CASE 1.—FIG. 1.



CASE 2.—FIG. 1.

Stained with Picrocarmine showing Hæmorrhages.



CASE I.—FIG. NO. 2 \times 115.

Photomicrograph, representing area (*p*) of Fig. No. 1. Thickened pia closely attached to the cortex.

- a.*—Blood-vessel with thickened wall, and filled with blood.
- b.*—Pia thickened and infiltrated.
- c.*—Cortex.
- d.*—Margin between cortex and pia.



CASE I.—FIG. NO. 3 \times 115.

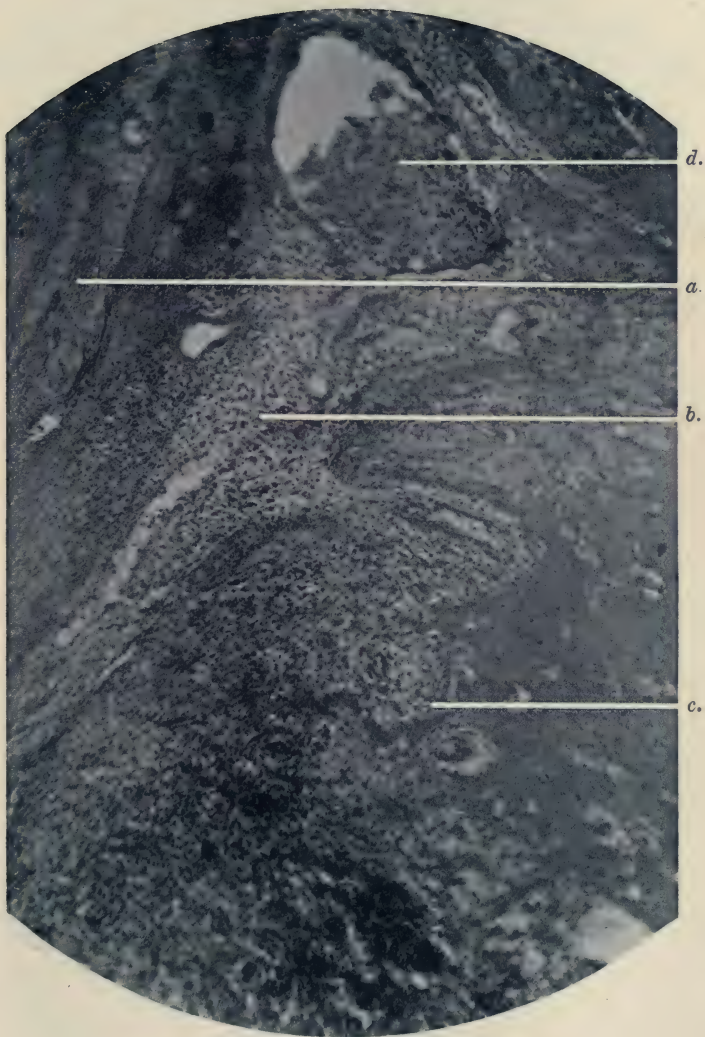
Photomicrograph, representing area (*p*) of Fig. No. 1. Pia with hyaline degeneration in capillary blood-vessels.

a.—Capillary vessels which have undergone hyaline degeneration.

b.—Disorganised cortex.

c.—Shrunk blood-vessel with large peri-vascular space.

d.—Same.



CASE I.—FIG. NO. 4 \times 325.

Photomicrograph, representing area (c) of Fig. No. 1. Disorganised cortex with pia closely attached.

a.—Connective tissue growth in pia.

b.—Cell infiltration between cortex and pia.

c.—Disorganised cortex.

d.—Dilated blood-vessel filled with blood.



CASE I.—FIG. NO. 5 \times 325.

Photomicrograph, representing area (*n*) of Fig. No. 1. Neuroglia tissue as seen in the neighbourhood of the area of softening.

a.—Neuroglia cells.

for by supposing that it occurred in the interval between the first and second operations. At one particular point, just opposite to where the connective tissue septa are seen extending into the cortex, there is a small group of capillary blood vessels which have undergone distinct hyaline degeneration (see fig. 3); in this particular portion of the pia we find the densest connective tissue formation (figs. 2 & 3).

Cortex.—The subpial zone is very much disorganised by the infiltration of spheroidal cells (fig. 4), and by septa of connective tissue which have grown down from the pia into the cortex. The blood vessels appear shrunk and some are entirely occluded, and the walls of the vessels are thickened and infiltrated with cells. The perivascular spaces are wider than normal. It is especially in the environs of the blood vessels that the acute process seems to have been most active. Just beneath this subpial area, is a zone of cortical tissue which on casual inspection appears entirely normal. At the junction of this zone with the white matter, is situated an oval-shaped area of softening which merges into the grey matter, while the neuroglia overgrowth is just beneath the softening (see fig. 1 S¹ S², and appears to pervade the entire mass. Surrounding and passing through the softened mass, many diseased blood vessels are seen. The softened area consists of degenerated and disintegrated nervous elements, and a number of bodies which closely resemble Gluge's corpuscles. The neuroglia mass fills up the greater part of the oval area, and is characteristic in every way of this peculiar tissue (see fig. 5).

Ganglion Cells.—Although a large number of the cells appear perfectly normal, many of them show distinct degenerative changes, these degenerative changes are especially seen in the protoplasm of the cells. A number of cells show a more or less granular nucleus, in others the nucleus appears to lie close to the limiting wall of the protoplasmic substance, while still in others no nucleus whatever can be seen. The protoplasmic substance is dotted here and there with empty-looking spaces, which very much resemble vesicles in appearance; in others there is apparently a roughened outline of the walls of the cell. A few cells have

the appearance of a simple wall with a nucleus, but no demonstrable protoplasmic substance. Some of the cells are distinctly larger than normal, probably swollen, while others are considerably smaller and shrunken in appearance. Small bodies resembling a nucleus with its nucleolus, are found scattered here and there amongst normal and degenerated cells. These are unquestionably the remains of degenerated cells, of which nothing remained except the nucleus and the nucleolus. The peri-cellular spaces around some of the cells are larger than they should be, in others the spaces appear well preserved and normal. These changes were present throughout the whole cortex, but were especially distinct in the layer of large pyramidal cells. The whole picture of this cell degeneration is that of a slow, degenerative process, combined with a more or less acute one. Whether an actual hyperplasia of the neuroglia exists in the cortex proper, could not be determined with any degree of certainty, although there were patches here and there which very closely resembled sclerosis.

To recapitulate. The important pathological findings in this case were:—

(1) *Meningo Encephalitis*.—This seems to have been chronic in character; to this there was added an acute exacerbation, which probably occurred in the interval between the first and second operations. Whether or not this chronic meningo-encephalitis had its origin in a trauma cannot be established as fact from what we observe in the specimens under consideration.

(2) *Marked Obliterative Changes in the Blood Vessels of the Pia and Cortex*.—This vascular depravity was associated with the formation of new capillaries which appear to have undergone hyaline degeneration.

(3) *Slow Degenerative Changes in the Ganglion Cells*.—The neuroglia hyperplasia in the cortical areas, although distinctly demonstrable, is not very marked in this tissue. Some acute degeneration was also present.

(4) *Softened Areas*.—These must have resulted from the obliteration of some of the blood vessels, just at the junction of the grey matter with the white, where the blood supply of the brain is poor.

(5) *Replacement of this Softened Area by true Neuroglia Tissue.*—This is well demonstrated in the specimen which shows both softened areas and neuroglia tissue closely associated together in such a way that surrounding each accession of softening there is an advancing zone of neuroglia tissue.

A number of hæmorrhages were noticed in the specimens, but as the blood appeared of comparatively recent occurrence, it is believed that they were produced during the operations.

CASE II.

This case, one of idiopathic focal epilepsy, I shall refer to in briefest manner, as the case will probably be reported in detail later by Dr. Dana, to whose clinic she came, and by Dr. Curtis, who did the operation.

The patient, a married woman, 30 years old, of good family and personal history, has suffered for six years from a convulsive affection, which she referred to as a cramp of the left leg, which is often associated with foaming at the mouth, biting of the tongue, involuntary evacuation of excrement, and periods of unconsciousness lasting from five to twenty minutes. The attacks have gradually got more frequent, and now she may have two distinct *hauts mal* attacks and twenty of the cramp conditions of the left leg in twenty-four hours.

Her description of the convulsive attack in the leg and the general seizure in connection with the mental and physical condition after an attack, the increasing frequency of the attacks, and the progressive mental enfeeblement, left no doubt as to the nature of the malady, viz., genuine epilepsy, the attacks of which always began with the same localised spasm.

The cortical area in which the left leg centre is situated was outlined, and a corresponding opening made in the skull. The dura was laid back, and the cortex excised two days later. The piece of brain tissue which was removed measured about 4 cm. in length and 2 cm. in thickness. This tissue was prepared for the reception of the Nissl and picro-carmin stains in the same way as the tissue from the first case.

Sections of the removed cortex prepared in this manner show distinct and, I believe, suggestive changes, particularly in the layer of large pyramidal cells. Without going into histological details, we may recapitulate the pathological findings in this case briefly as follows:—

The shape of the section of the cortex shows that the excised piece was from the bottom of a fissure, and only a small portion of the lateral walls of the fissure are included (fig. 1).

The most striking feature on first examination of the specimens, is the large number of hæmorrhages scattered throughout the entire section, but particularly conspicuous in the deeper parts of the cortex, that is near the line of section. The crimson colour of these hæmorrhagic spots on the cut surface of the specimen indicates distinctly their recent origin. They were, doubtless, due to the trauma incident to the operation. The specimen was extremely hard, and especial precaution was required to cut the sections even with the paraffin imbedding. This excessive hardness was probably due to these hæmorrhages, as nothing else could be found to account for it.

The specimen stained with picro-carminé gives the best general outline of the specimen, and shows the distribution and character of the hæmorrhages (fig. 1). In keeping with the traumatism of the operation the largest patches of hæmorrhage are in the neighbourhood of the line of excision. Towards the surface of the cortex they become smaller, and leave a larger amount of intact tissue between them in the deeper regions. In the superficial layers their size varies from a minute point to a size sufficient to be seen with the naked eye. The larger patches show distinct agglomerations of red blood corpuscles so dense as to almost obscure any other tissue within the affected area. In other places the corpuscles are quite discoloured, and form such a thin and clarified layer that they are hardly seen. In such spots the neuroglia meshes stand out distinctly. In many places only neuroglia meshes are seen. The blood corpuscles and nerve cells have evidently fallen out in this region. This explanation is supported by the fact that all sorts of

transitions of the conditions described are met with. One also sees occasionally a blood vessel within a nearly complete gap of tissue. It is extremely improbable that these gaps are peri-vascular spaces, in view of the fact that in some places blood vessels are to be seen in the middle of patches of completely rarified neuroglia meshes, as well as in the centre of distinctly hæmorrhagic spots.

The multiplicity of hæmorrhages makes it difficult to say whether or not there are distinct vascular and interstitial changes other than those referred to.

Nerve cells. (Figs. 2 and 3).

Specimens prepared after the Nissl method. The superficial layers of cells including the small pyramids down to the layer of large pyramids appear to be perfectly normal. The large pyramids, however, present striking changes. In the first place, there is a great scarcity of these cells (fig. 2). This may be accounted for, in part, by the fact, that at this depth the hæmorrhages occupy a great portion of the tissue, but it is to be remarked that the cells that are found, which by their position and shape correspond to the large pyramids, are for the most part intensely altered, they present the characteristics of atrophy; they are thin, shrunken, with attenuated spiral processes; they stain much more deeply than normal cells, or, at least, they refuse to part with the stain in the differentiation fluid, and they have lost the chromatine striped structure of normal cells. The nuclei which stand out so distinctly in normal cells are here scarcely to be differentiated from the cell body, for it is nearly as dark as the latter. In many cells, indeed, no nuclear structures can be made out. Only a few of the large pyramidal cells met with, present a normal appearance.

The hæmorrhages in these specimens have been described in considerable detail, not because they have, I believe, any particular pathological significance, but to show how numerous and severe hæmorrhages into the substance of the cortex may be after such operations. Particularly in cases where excision of the cortex is done some days subsequently to opening the skull.

It is probable that the blood which formed thin, white,

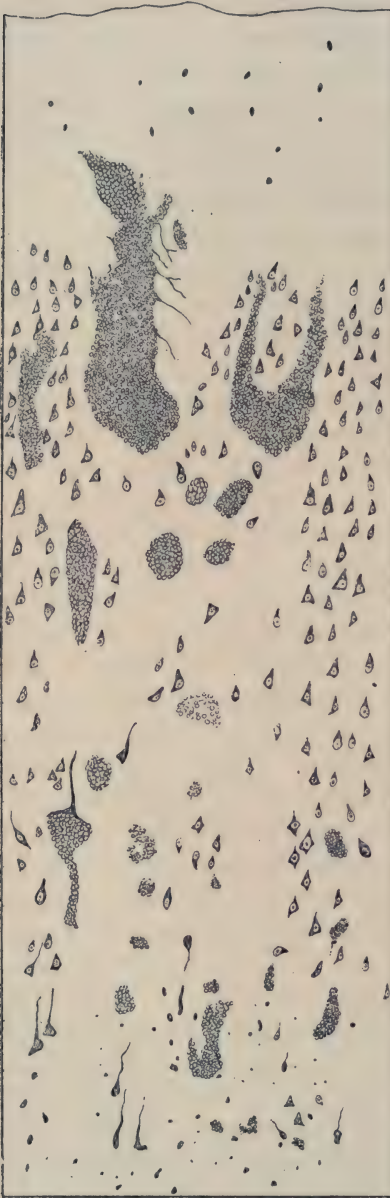


FIG. 2.

Showing scarcity and degeneration of the layer of large pyramidal cells.



FIG. 3.

Individual cells form the layer of large pyramids.

clarified layers dates from the first operation, and the larger more recent looking areas from the second.

The changes in the ganglion cells of the large pyramids we do not believe are secondary to the hæmorrhages, although the scarcity of cells is, undoubtedly, due in part to them. That such striking changes of degeneration could go on in these cells in two days, the time elapsing between the first and second operations, seems scarcely possible.

If these operations should receive corroboration by others, they would indicate not only the direction in which the pathology of epilepsy lies, but the advisability of operation in such cases.

The pathological findings in the first case would seem to be not difficult of interpretation; a patient with Jacksonian epilepsy, has a distinct and localised spasm, which points to a lesion of the cortical centre for the right hand. There are evidences that this lesion has existed sufficiently long to cause beginning secondary changes. The hand centre on the surface of the brain is located and excised. After the acute accompaniments of the operation have passed off, the patient remains free for a year of any attacks; and the only ill results of the operation are the natural sequences, which attend the removal of a part of the motorial area, namely, partial motor disability. Microscopical examination demonstrates that in the piece of cortical tissue which was removed, there are definite pathological conditions which may be postulated to explain the clinical symptoms from which the patient suffered. Logically, the removal of the focus of disease should be followed by a cessation of symptoms, that is, cure of the disease.

The two different lesions that were found in the diseased area may be explained by considering the meningo-encephalitis, in all probability the original one. The second set of lesions, the degenerative changes in the ganglionic cells, and the neuroglia-hyperplasia in the cortical areas were secondary to the meningo-encephalitis, and immediately sequential to the vascular obliterative changes in the blood vessels of the pia and the cortex.

The other changes, the hæmorrhages and the diapedesis

immediately about them are probably in no way a part of the patient's disease, but were directly due to the first and second operations.

It is believed, therefore, that the epilepsy in this case was due to the lesions which are herewith demonstrated; that the diseased area in the patient's brain was entirely removed, and that by virtue of such removal the attacks have not recurred; and furthermore by virtue of the very early operative interference, before the motor ganglion cells in the surrounding cortex had become interfered with in their nutrition, or perverted in their function, that the disease will not recur.

We think it advisable not to advance any opinion based on knowledge obtained by the study of the clinical and pathological aspects of these cases, as to what the pathology of so-called idiopathic epilepsy may be, although there is temptation to do so. We are so keenly alive, however, to the liability of error in generalizing from individual cases, that we entirely refrain. We believe, however, that the findings in these cases advance our knowledge of the pathology of epilepsy, insomuch as they show that in the very early stages of that disease there are changes in the sources of motorial impulses, the ganglion cells of the cortex, which can be demonstrated by modern technic.

In closing, it may be said that these cases are not reported as cured, although the first patient has been free from epileptic manifestations for more than a year. The time which has elapsed since operation on the second patient is too short to make statement regarding its efficiency justifiable, although the patient has made gratifying progress since the operation.

If these cases tend to demonstrate anything, it is that there are changes in the brain structure in cases of epilepsy which conscientious surgeons consent to operate on. Therefore it may be asked why should not such diseased tissue be removed in every case? If it be removed, we believe that this operation for epilepsy will not pass into complete desuetude, although it will ever have an extremely narrow field of usefulness.

MIRROR WRITING.

BY F. J. ALLEN, M.D.CANTAB.

Professor of Physiology in Mason College, Birmingham.

A STUDY of the subjective phenomena of left-handed or mirror writing may help to throw light on the nerve processes concerned in writing and other complex co-ordinated actions. The faculty of mirror writing is probably not at all rare. It may be possessed more or less by most persons, but may remain unobserved. It is apt to be discovered for the first time when some nerve disease renders normal writing difficult or impossible; the patient then falls back on the resource of left-handed writing. Thus mirror writing is often a symptom of nerve disease; but the disease need not be the cause of the existence of the faculty, but only the cause of its discovery. At all events in my own person the faculty of mirror writing has existed from childhood. I discovered it accidentally about my thirteenth year, when, on making the first attempt, I was able to write backwards with the left hand without error or hesitation. Ever since that time I have been able to write left-handed without practice.

The most noticeable subjective phenomena are the following:—

(1) It is very easy to write in mirror fashion with chalk on a black board, co-ordination seeming to be perfect in the movements of the left arm and wrist. But when using a pen with the left hand, there is a slight difficulty in some of the movements. There is (in the latter case) no hesitation as to the formation of the letters; but occasional awkward movements will produce distorted lines, and there is special difficulty in keeping up a smooth onward movement, “the movement of alignment.”

(2) Writing is only a little slower with the left hand than with the right. In the fastest possible mirror writing there is no error in the form of the letters—only such want of neatness and distinctness as occurs in writing too fast in the ordinary way.

(3) The following actions are quite easy :—

(a) Writing the same thing simultaneously with both hands.

(b) Writing backwards with the left foot, *i.e.*, scratching on sandy ground, as one sometimes does with the right foot.

(c) Using the deaf and dumb finger alphabet in the reversed or left-handed manner.

(4) If I learn a new alphabet with the right hand, I can write it with the left; it is not necessary to learn it separately with the left hand; the nervous system, after being trained to any kind of writing with the right hand, is able to send its co-ordinated messages almost equally well to either hand.

(5) The assistance of sight is not necessary in mirror writing. Indeed, sight may even impede it: and although it is easy to write left-handed, it is difficult to read what one has written. When I look at my own mirror writing, it sometimes conveys no meaning to me, and I experience sensations which are probably much the same as those of a person who is word-blind, but retains the faculty of writing. In order to decipher such writing, it is necessary to run the eye along the lines, and so reproduce the sensations which accompany the act of writing. I find considerable difficulty in reading print reversed by means of a mirror, but hardly any difficulty in reading a book held upside down.

It may be necessary to state that I have no tendency to be left-handed, though I might almost be called ambidexter.

The sensations accompanying all kinds of writing, whether with right hand or left hand, right foot or left foot, are so similar as to suggest that in all cases the messages start from the same region of the brain; but it seems as if a series of commutators existed at a lower level,

whereby the impulses could be turned into different channels leading to analogous but sometimes heteronymous groups of muscles. This supports the view that the true graphic centre is not coincident with either of the motor centres, but superior to all of them.

I append some words written both in mirror and in ordinary writing; the features may be compared with the assistance of a mirror.

position - mirror
Mirror - writing

FIG. 1.

Broca's Convolution
Broca's Convolution

FIG. 2.

Journal of Neurology
Journal of Neurology

FIG. 3.

Critical Digest.

ABSTRACTS OF PAPERS ON THE ETIOLOGY AND PATHOLOGY OF DISSEMINATED SCLEROSIS.

Etiology.

MARIE (1), in discussing the etiology of the disease, points out that the most common age at which the symptoms commence is the early part of adult life (20-30), and he regards this as a point of some importance in the diagnosis. In any case of doubtful diagnosis, disseminated sclerosis may be almost eliminated, if the disease has commenced after the 40th year. On the other hand, however, the disease is occasionally met with in infants. Marie believes that infectious diseases are the most important factors in the causation of disseminated sclerosis. When one considers the number of facts in favour of this relationship published by various authors, and the number of cases in which the affection of the nervous system has first shown itself in the course of the infectious disease or during convalescence therefrom, Marie thinks it is impossible to deny that there exists a relation of cause and effect.

As regards the nature of these infectious diseases, Marie found, in twenty-five cases which he collected, that typhoid fever was the antecedent in eleven. Pneumonia, malaria, measles, scarlet fever, and especially small-pox, have been followed by disseminated sclerosis. Other infectious diseases—diphtheria, whooping cough, erysipelas, dysentery, and cholera—have occasionally been recorded as antecedent in disseminated sclerosis. To this list Marie adds "unknown infectious diseases." He discusses the relation of disseminated sclerosis to the micro-organisms of infectious diseases. If these micro-organisms were the cause, one would expect to meet with disseminated sclerosis much more frequently in the course of various infectious diseases. Hence he regards a combined or mixed infection, which is so frequent in the course of the infectious diseases as the exciting cause. Though it is not possible, at present, to come to any definite conclusion as to the exact pathogenesis, still Marie thinks it is a well-established fact, that disseminated sclerosis stands in close relation, as regards causation, with the infectious diseases.

Totzke(2), in his inaugural dissertation on disseminated sclerosis in childhood, quotes cases, published by Leuch, of a mother and child, who both suffered from the disease. The mother was suffering from the disease during her pregnancy; the child was healthy until the 7th year; then symptoms of disseminated sclerosis appeared, and the disease terminated fatally. From a review of 35 cases in children, 33 of which were collected from medical literature, Totzke regards infectious diseases as the most important factor in the etiology. In these 35 cases the disease followed some acute infectious ailment in 13—diphtheria, 4; scarlet fever, 4; measles, 1; whooping-cough, 2; cholera nostras, 1; unknown febrile affection, 1. The author mentions two instances, in which two children of the same parents suffered from disseminated sclerosis.

In 31 cases of the disease in childhood, the onset was before the 6th year of age in 19 cases; in 12, between the ages of 6 and 14 years. In 2 cases the symptoms appear to have been present from birth. In disseminated sclerosis in the adult, the commencement of the disease can sometimes be traced to childhood. In some cases the patient has presented typical symptoms in childhood; in other cases certain symptoms only have been present.

A case of disseminated sclerosis following influenza is reported by Massalongo (3). The writer has met with a case also, in which the disease followed influenza. But considering the prevalence of influenza during the last few years, more evidence is required before any conclusion can be drawn with regard to the connection of the two affections.

Torti and Angelini (4) record two cases of chronic malaria, in which symptoms resembling those of disseminated sclerosis developed. The first patient was a man aged 21 years, who, after suffering for three months from malarial fever of an irregular type, began to be troubled with vertigo and vomiting. Then weakness of the legs, slow scanning speech, increase of the knee-jerks, slight nystagmus, were noted. Examination of the blood revealed the malarial parasites. Under treatment, improvement occurred, and the malarial parasites disappeared from the blood. The patient was discharged from the hospital, but in a short time a return of fever occurred, and in a few days the following symptoms developed: spastic paresis, ataxia of the limbs, nystagmus, tremor, increase of the reflexes, scanning speech, headache, and vertigo. The malarial parasites were again found in the blood. The symptoms again subsided, and in two months

the patient was discharged well. The second case was a man aged 22. In August and September, 1890, he had suffered from malaria. In November he presented the following symptoms: headache, vertigo, scanning speech, nystagmus, intention tremor, increase of the reflexes. The malarial parasites were present in the blood. After three weeks' treatment he recovered.

Cases of multiple sclerosis after malaria have been recorded by Camellis, Boinet and Salebert (pseudo-sclerosis), and Bignamic Bastianelli. Clinically, three forms of sclerosis are met with after malaria. (1) Symptoms of multiple sclerosis may occur temporarily with severe attacks of malarial fever. (2) Symptoms of multiple sclerosis (the duration of which varies) may occur after the fever. (3) Symptoms of sclerosis may occur suddenly with fever under the form of very acute malarial attacks. All three forms show a tendency to improvement and recovery. Torti and Angelini believe that the cause of these symptoms is a poisoning of the nerve centres (not a vascular disease), and refer to a case of pernicious malaria, recorded by Marchiafara, with bulbar symptoms (paralysis of the facial, hypo-glossal and vagus) in which the vessels in the region of the bulbar nuclei were filled with blood cells containing parasites (parasitic thrombosis).

Jordan (5) reports a case, under the care of Suckling, in which symptoms of disseminated sclerosis came on suddenly a few days after a sudden and violent mental shock.

Leyden (6) states that cases, beginning like an acute myelitis, sometimes pass on to sclerosis. Disseminated myelitis may follow an acute illness and *post-mortem* examination, after the nervous symptoms have existed for many years, may reveal multiple sclerosis.

In many cases of spinal paralysis which present the symptoms of acute myelitis, the autopsy made after the disease has existed for many years, has revealed sclerosis of the spinal cord.

Pathology.

Charcot, in his earlier work on the pathological anatomy of disseminated sclerosis, drew attention to the dilatation of the blood vessels in diseased patches and to the thickening of their walls, especially of the external coat. Charcot's view as to the interstitial origin of the sclerosis has been accepted by most pathologists, but not by all.

Huber (7) has quite recently reported a case of disseminated sclerosis, in which, in some parts, the diseased patches presented

simply disappearance of nerve fibres without changes in the interstitial tissue, or without changes sufficient to cause the destruction of the nerve fibres. Also in the patches the vessels were not changed, and showed no relation to the distribution of the diseased process. In these patches the author regards the disease as a primary affection of the parenchyma. In other patches there was marked sclerosis of the interstitial tissue, and between the two kinds of patches there was no sharp distinction. Hence the author believes that the patches belong to one diseased process, in which an affection of the parenchyma is the primary change, and the sclerosis secondary. Further, in longitudinal sections of sclerotic patches, sometimes small linear processes were seen, which were situated exactly in the course of the atrophied nerve fibres, connective tissue taking the place of degenerated nerve fibres. If the changes proceeded primarily from the interstitial tissue, one would expect the new formed neuroglia to be equally developed laterally at these parts.

The vascular origin of the patches in disseminated sclerosis was suggested by Rindfleisch many years ago. More recently, Ribbert (8) from a study of the microscopical changes, has arrived at the conclusion that disseminated sclerosis is related to vascular lesions, and due to the presence of some irritating agent in the vessels. In the case which he reports, both greyish-red, soft, recent patches, and old grey patches, were found at the autopsy. In the former, a large, greatly distended blood vessel was found running through the centre of each. Around the vessels in these patches there was a collection of round cells. In two places he found partial obstruction of the lumen of an artery by a peripheral thrombus, consisting of white blood corpuscles.

Ribbert believes that some irritating substance is distributed by the vessels to the central nervous system. Under the influence of this irritating agent a fibrinous clot forms at one part of a small vessel. The clot generally occupies only a portion of the periphery of the lumen of the vessel. At this point an irritation of the vessel wall occurs, and more or less marked diapedesis of lymph corpuscles follows. A peri-vascular inflammation occurs, which extends in a concentric manner around the vessel from which it commenced. The exact nature of this irritating agent remains unknown.

Bastian (9) states that on several occasions he has found the larger vessels of a patch of sclerosis blocked by old and firm thrombi.

Marie (10) draws attention to the vascular changes in dis-

seminated sclerosis. In the centre of a patch of sclerosis a dilated blood vessel is often found, and the sclerotic patch appears to have developed around the vessel. Considering the relation of disseminated sclerosis to infectious diseases this localisation is not surprising. Marie points out two interesting facts with regard to the patches of sclerosis. One is, that the periphery of many patches presents more or less numerous granular corpuscles, which indicate that the morbid process is still active. A second interesting fact is that in some old standing cases, at the autopsy, in addition to old patches of sclerosis, there are small patches containing granular corpuscles both at the centre and the periphery—in other words, recent patches of disease. The first fact above mentioned indicates that the morbid agent remains in the patch and continues its action in an almost indefinite manner. The second fact shows that the morbid agent persisting in the organism is able to diffuse itself and to cause the development of new patches of sclerosis. Though our definite knowledge as to the causation of disseminated sclerosis is so slight, Marie thinks, however, we may accept the following conclusions: disseminated sclerosis is a sclerosis of vascular origin, the general aspect of which appears to indicate a process allied to *embolism*: it arises under the influence of various infectious diseases, probably by the mechanism of combined infection.

Hess (11) describes the pathological changes in a case of disseminated sclerosis, drawing attention to the persistence of axis-cylinder in the sclerotic patches and to the absence of secondary degeneration. In addition to the patches of firm sclerosis, both in the medulla and cord, areas of diffuse neuroglia proliferation were met with, in which the medullary sheaths of the nerve fibres were simply diminished and not absent. The condition of the vessels varied in different parts. Many small vessels, especially the capillaries, were normal. At some parts the walls were thickened, chiefly in the larger vessels; or the vessels were increased in number; or they were dilated; in other parts the lumen was diminished. At some parts the lumen was completely obliterated by thickening of the vessel walls. The peri-vascular lymph sheaths were distended with round cells and the nuclei of the vessel walls increased.

In addition to the patches presenting the usual appearances of disseminated sclerosis, there were in the pons, medulla, and at one part of the lumbar region of the cord, areas in which there was marked cell infiltration. These patches the author regards as the results of circulatory changes and as an early stage in the development of disseminated sclerosis.

Hess believes that the sclerosed patches commence with marked disease of the vessel walls. In the patches of diffuse sclerosis, he believes that the nutrient vessels are only so far diseased that an emigration of white blood corpuscles and an increase and new formation of the neuroglia occurs, analogous to the increased interstitial tissue in the disturbance of nutrition in other organs. Hence only here and there does the myelin of the nerve fibres disappear. In other vessels supplying sclerosed areas as the changes in the walls are more marked. As a result the neuroglia is increased and the white substance of the nerve fibres disappears owing to defective nutrition. Only after a long period of disturbance of nutrition does the axis-cylinder disappear. The changes in the vessel vary in degree. They are, at some parts, only slight and allow the emigration of white corpuscles to occur. In other places the changes are marked and may lead to complete obliteration with severe nutritional disturbances in the region of distribution; between both these extremes there are various modifications.

By obliteration of the vessels (owing to thickening of the walls) the same circulatory disturbances would be produced as by the peripheral thrombi observed by Ribbert. The author looks upon disseminated sclerosis as a process in which the myelin of the nerve fibres disappears and the neuroglia proliferates owing to primary changes in the vessels.

Popoff (12), from the microscopical examination of the central nervous system in four cases, comes to the conclusion that the usually accepted views as to the pathology of disseminated sclerosis are erroneous. He recognises two forms of the disease—sub-acute and chronic. He does not believe that there is an increase of the connective tissue in the sclerosed patches, but thinks that what most observers have looked upon as tracts of connective tissue, lying between the nerve fibres, are only degeneration products of the nerve fibres themselves. He regards the methods of staining, which have been usually employed, as unsuitable for studying the nature of the tissue which replaces the nerve fibres in the sclerosed patches. The vessels, which form the centre of every patch, must be looked upon as the starting point of the affection. The changes in the surrounding tissue spread gradually from the central vessel, and this gradual advance of the process is especially marked in the chronic cases. All the tissues around the vessels are finally affected, but not at the same time.

The chief change in the vessel wall consists in cellular infil-

tration, followed by thickening, and sometimes concentric narrowing of the lumen. Also an abundant emigration of leucocytes occurs into the surrounding tissue. The leucocytes do not increase in size; they do not become changed into connective tissue, but degeneration. Through their presence between the nerve fibres probably they exert an injurious influence on the vitality of the fibres, but they are not the chief cause of the metamorphosis.

The sub-acute form of multiple sclerosis differs from the chronic in this, that in the former the cell infiltration of the vessel walls is more irregular than in the chronic form; the number of leucocytes is distinctly greater, and the course is more acute. In some place the author has found what he regards as regenerated axis-cylinders, and he believes that the great numbers of axis-cylinders without medullary sheaths, which have been described by Charcot, Leuker, Hess, and others, in sclerosed patches, are to be regarded as new-formed axis-cylinders—regenerated axis-cylinders.

At the periphery of a sclerosed patch a new formation of blood vessels occurs—a condition which the author regards as favourable to the regeneration of axis-cylinders of nerve fibres.

According to Popoff, the generally accepted views as to the pathology are false, because—

- (1) No proliferation of connective tissue occurs in this disease.
- (2) Not only do the nerve fibres lose their medullary sheaths, but the axis-cylinders also degenerate.

The pathological process consists in a gradual degeneration of the whole tissue surrounding an affected vessel. Besides the degenerative process, a regeneration of axis-cylinders occurs, perhaps only in certain parts of the cord (the pyramidal tracts and Goll's columns).

Buss (14) reports a case which during life presented symptoms of spastic spinal paralysis, but the autopsy revealed disseminated sclerosis, with secondary degeneration (ascending and descending) in the spinal cord. In many places the axis-cylinders were destroyed. Peri-vascular cell infiltration was marked, and the walls of the vessels were thickened. In the brain there were numerous small patches; in the centre of the patches a small vessel was seen on section. These vessels were veins with thickened walls, which were surrounded by cell infiltration. Buss regards the vascular changes as the starting point of the lesion.

The clinical history of the case is unusual, from the fact

that there were no tremors on voluntary movement and no scanning speech. The first symptom was dull pain in the back. A few weeks later, after severe exposure to cold and wet, the patient suffered from a marked rigor, accompanied by vomiting and pains and tremors in the limbs. After this attack the nervous symptoms became much worse. The same acute symptoms followed a large dose of aloes (forty-one pills taken one after the other), and afterwards the spinal symptoms again rapidly became worse. Whilst the patient was in the clinic these symptoms—marked rigors with high temperature, &c.—occurred several times. In the attacks the urine became dark, and contained blood pigment. After one of these attacks of rigor, with febrile symptoms and hæmoglobinuria, the spinal symptoms for a third time became rapidly worse, and an acute bed-sore developed. Buss believes that the rapid advance of spinal symptoms, after the large doses of aloes, was due to blood dissolution. Aloes, in toxic doses, may give rise to necrosis of the renal epithelium and hæmoglobinuria. Small doses of substances producing hæmoglobinuria have been found to produce thrombosis in the small vessels of the heart, the lungs, and in the small veins in other organs. Buss found in his case of disseminated sclerosis that the sclerotic patches were grouped, almost without exception, around small veins, but no thrombi were detected.

Buss thinks that in several points there is a strong resemblance between an attack of hæmoglobinuria and the effect of exposure to cold. There are several facts which render it probable that the acute symptoms following the exposure to cold and wet in the above-mentioned case were due to hæmoglobinuria. The author believes that (1) after the exposure to cold; (2) after the aloes pills; (3) after the attacks of hæmoglobinuria, similar general blood conditions occurred; on all these occasions the spinal symptoms suddenly advanced markedly.

Afariassiew and others have shown that in hæmoglobinuria, produced experimentally, interstitial changes are found in the various organs. They attribute the changes to the irritating (or inflammation producing) properties of substances formed by blood dissolution. Buss believes that in consequence of blood changes (dissolution) some substance is produced which has an inflammatory exciting influence on the small veins of the nervous system. Changes are produced in the walls of the veins, and secondary changes occur in the nervous system around. This he looks upon as one cause only of disseminated sclerosis. Many patients refer the disease to a severe cold (taking cold),

and Buss thinks that blood changes may be produced thereby, and a hæmoglobinæmia produced without hæmoglobinuria, and this may be an explanation of the apparent influence of cold in producing the disease in some cases.

Taylor (15) gives a detailed account of the pathological changes in three cases of disseminated sclerosis. In the first case, the whole cross section of the spinal cord at many regions was degenerated. Often only a small number of fibres which still retained their white substance, were recognised on a transverse section. Both grey and white substance were affected indifferently. The extra medullary parts of the anterior and posterior roots were degenerated at some parts. The nuclei of all the cranial nerves (in the medulla, pons, and region of the corpora quadrigemina) were affected. The central canal of the lower part of the medulla, the whole floor of the fourth ventricle, and the aqueduct of Sylvius up to the oculo-motor region were involved in one continuous patch of sclerosis.

In the second case, it is worthy of note that the cauda equina, the spinal roots, and the cortex of the cerebellum were affected: and in the third case, the internal capsule, cortex of the cerebrum and cerebellum were affected.

In opposition to the views of previous writers, Taylor concludes from his own observations that the white and grey substances are affected irregularly; that there is no prædilection spot in the central nervous system for the patches of sclerosis; that neither the cortex of the cerebrum nor cerebellum are spared.

In two of Taylor's cases all the cerebral nerves were affected (except the olfactory) at some part; either at the nucleus, or in the central or peripheral course of the nerve.

The optic chiasma was also the seat of sclerosis in all of Taylor's cases.

The author confirms the observations of Charcot and Erb, that the spinal nerve roots are often affected; he also found well-marked degeneration of the nerve fibres of the cauda equina (as above mentioned).

According to Charcot, at the periphery of the patches of sclerosis there is a transitional zone, of less marked degeneration before the healthy nerve tissue is reached. But Taylor has observed many sclerosed patches, which were sharply defined, and in which there was no transitional zone of partial degeneration, a patch of marked sclerosis, in which medullated nerve fibres were absent, being bounded by healthy nerve tissue.

Taylor has also found complete atrophy of the medullary sheaths of the nerve fibres, both at the centre and at the periphery of the patches. In some patches the whole area affected, on microscopical examination, presented the appearance which has been described as that of the terminal stage.

The author refers to the absence of secondary degeneration in disseminated sclerosis, which has been so frequently noted; but he admits the possibility of such changes when the axis-cylinders are completely destroyed in a sclerosed patch, though he believes a well-marked secondary degeneration of the various tracts to be exceedingly rare.

Taylor's observations show that degeneration of ganglion cells only occurs in an advanced stage of the process, and not directly the grey anterior horn is implicated, as is stated by some observers. Undoubtedly the cells persist for a long time, and, for the most part, are in a state of functional activity. This is the reason why the reaction of degeneration and muscular atrophy are so rare in this disease. The first change in the cells consists in marked pigmentation. In the normal spinal cord pigmentation is observed, but not to such an extent, and in such young individuals, as in disseminated sclerosis. A collection of pigment which fills the whole cells does not occur under normal conditions. A further degeneration, up to complete atrophy of the cell, only rarely occurs, and that in the last stages of the disease.

The changes in the vessels in Taylor's cases were not marked. In one case the small vessels, especially the capillaries, were enormously increased in some regions. They were filled with blood; here and there were small hæmorrhages, and often extravasated white blood corpuscles. Some vessels showed moderate thickening of the walls, with a minimal increase of nuclei. The peri-vascular spaces were often dilated.

Similar changes, but less marked, were found in the second case. In the third case, scarcely any abnormality of the vessels could be detected. In the first two cases, the vascular changes were most marked at the floor of the fourth ventricle, where the sclerotic degeneration was very extensive. Taylor, however, is of opinion that the vascular changes were not the starting point of the sclerosis for the following reasons:—

- (1) Because the patches were not always distributed in relation to the vessels.

- (2) Because in markedly degenerated parts, often the vessels showed no essential changes.

(3) Because in one case vascular changes were absent.

[It is to be remembered, however, that in Taylor's cases the pathological examinations were made at a comparatively late stage of the disease.]

A case reported by the writer (16) illustrates several interesting points with regard to the pathology of disseminated sclerosis. The course of the disease was very rapid. Indefinite symptoms were noticed twelve months before death, but distinct symptoms of the disease only appeared within the last five months of life. On pathological examination, in addition to old disseminated patches in the brain and cord, presenting the usual naked eye and microscopical appearances of multiple sclerosis, there were four *very soft* cerebral patches. The largest was in the right temporo-sphenoidal lobe ; it was greyish-yellow in colour, almost diffuent, and from the cut surface a small quantity of turbid greyish fluid escaped. There were other cerebral patches intermediate in consistence between these soft cerebral patches and the firm sclerosis patches in the cord. Sections of the softened patches, even at the centre, were seen to consist almost entirely of round cells, compound fat granular cells, and dilated blood vessels, the peri-vascular sheaths of which were distended with leucocytes. The appearance closely resembled that of acute cerebral softening. In the firmer patches there was increased fibrous connective tissue, and the usual microscopical appearance of sclerosis. A striking feature in nearly all of the patches, was the marked dilatation of the blood vessels, and the distension of the lymph sheath with leucocytes. In the upper cervical region was a recent wedge-shaped patch, in the region of the anterior median fissure. This area was densely infiltrated with leucocytes. The vessels in the fissure were greatly dilated and the peri-vascular lymph sheaths distended with leucocytes.

At the commencement of the anterior median fissure (*i.e.*, near the surface of the cord) in a number of sections there was a thrombosed vessel. In some of the sections taken from this part of the cord, in addition to the thrombus in the anterior median vessel, there were several small thrombosed veins in the pia mater on the anterior surface of the cord.

These thrombi are of interest in connection with the above-mentioned views of Ribbert and Marie, and the observations of Bastian and Ribbert.

Demange (17) reports an interesting case of sclerosis of the cord of vascular origin. During life the symptoms resembled those of amyotrophic lateral sclerosis. At the autopsy extensive athe-

roma of the vessels was found. The microscopical examination revealed disseminated sclerosis in all regions of the spinal cord. These patches were scattered most irregularly in the white matter, and in some parts penetrated the grey substance. The margins of the patches were not sharply defined, but were furnished with radiating processes (sclérose "non en plaques mais en taches"). In the centre of each small area of sclerosis was an altered artery, from which the sclerosis extended into the meshes of the neuroglia. The neuroglia was thickened and possessed numerous spider cells. The arteries appeared generally increased in number, and of unusual size. The endothelium was proliferated, the walls thickened, infiltrated with nuclei, and the lymph spaces filled with leucocytes. Demange regards the case as one commencing in a diffuse myelitis, proceeding from the periarteritis of the vessels—an interstitial, diffuse, and peri-vascular myelitis. He divides scleroses of vascular origin into two varieties (1) the disseminated sclerosis in patches; (2) certain interstitial diffuse scleroses, some cases being due to syphilis, others being connected with general atheroma of the vessels.

Demange (18) also reports two other cases with similar arterial changes. From the changes in those three cases he concludes: That the vessels of the cord may be affected by a peri-arterial sclerosis, connected with general atheroma. This lesion gives rise to spots of diffuse sclerosis (of vascular origin) and also to miliary hæmorrhages. Clinically the symptoms are those of sclerosis of the lateral columns of the cord.

Déjérine (19) thinks that it is more than probable, that the non-symmetrical scleroses of the spinal cord are all of vascular origin.

Hence, though the pathology of the disseminated sclerosis is as yet very obscure, a number of interesting points have, nevertheless, been made out.

(1) The distribution of the sclerosed patches in the most irregular manner throughout the nervous system, without any relation to nerve tracts or other nervous structures, whilst adjacent parts of the nervous system are normal, favours the view that the sclerosed patches are probably related to the distribution and lesions of the blood vessels.

(2) It is probable that some cases of disseminated sclerosis commence as myelitis or encephalitis.

(3) At the periphery of many patches of sclerosis, more or less numerous compound granular cells are found, with indications that the morbid process is still active. Also, in some cases at

the autopsy, in addition to old firm patches of sclerosis, there are soft patches containing compound granular cells both at the centre and at the periphery, *i.e.*, patches of recent development. These cases show that the morbid agent persists in the organism and is able to diffuse itself, and cause the development of new patches of the disease.

(4) Numerous observers have pointed out that in the earlier stages of the development a small artery is found in the centre of each sclerotic patch, and the chief changes are found just around the vessel.

(5) In a large number of cases distinct vascular and peri-vascular changes have been found, especially in the early stages of the development of a patch of sclerosis. In several cases thrombi have been found in a few vessels. These changes are very suggestive that the disease is associated with vascular lesions, either in the nature of a partial or complete vascular obstruction (multiple thrombosis of minute vessels), or of a process *allied* to multiple embolism (Ribbert and Marie), or possibly the presence of some irritating agent in the circulation may stimulate the endothelium cells of the walls of the vessels, and give rise to extravasation of lymph cells, and peri-vascular changes at certain spots, and as a result patches of degeneration may follow.

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Reviews and Abstracts.

Studien über Hysterie. Von Dr. Jos. BREUER und Dr. SIGM. FREUD, in Wein. Leipzig und Wein. Franz Deuticke, 1895, pp. 268.

THESE "Studies" form an original and valuable contribution to the theory of hysteria. The authors, in 1893 and 1894, published short preliminary communications of their observations on hysteria, and they now give in this book a full statement of their views. In BRAIN, vol. xii., pp. 125, *seq.*, will be found an abstract of their first papers. Briefly they pointed out in these papers the importance of ascertaining the exciting cause of hysterical phenomena, and the circumstances attending their first appearance; the influence in producing hysteria of "psychical" shock, and of violent emotional disturbances, for which there is no outlet: that on a "reaction," or outlet, for these pent-up emotions being afforded through the agency of speech, relief to the symptoms could often be given; and they described the existence of "hypnoid states" in hysteria.

In the former communications there was no room for cases, but in this volume five selected cases are given at length in order to show their method of procedure, and the nature of the evidence on which their views are based.

The essential particulars in the analysis of one case by Dr. Freud may be given. The patient, a young lady, aged 21, had suffered during the previous three years from family troubles; she had nursed her father through a long and painful illness, which ended fatally; then she nursed her mother through a severe ocular affection, necessitating a long stay in darkened rooms; and after that she lost a favourite sister, who died somewhat suddenly after a short illness. She complained of difficulty in walking, on account of pains, which were increased by walking. There was some tenderness of the skin and muscles of both legs, and an imperfect hysterogenic zone on the outer aspect of the right thigh. The history of common emotional disturbance afforded no explanation

why the patient should suffer from hysteria, nor why it should take the form of pain on walking and on movement of the legs. The first origin of the pain possibly originated whilst she was nursing her father, from jumping out of bed with bare feet to attend upon him. At this time she had also a love affair, and there was a conflict between her inclinations and her duty to her father. The hysterogenic zone on the thigh corresponded in position to the spot on which she supported her father whilst the dressings were being changed. Further examination under partial hypnosis elicited the fact that pain on walking had been first felt after a long walk with her sister's husband, during which she discovered that she regarded him with more tender feelings than she should do. Moreover, pains in walking, standing, or lying down seemed to be connected in memory with painful scenes in which she had been standing, walking, or lying respectively. The first period of her illness could be described as a monosymptomatic hysteria, *i.e.*, pains in one thigh connected with the strain of nursing her father; the second period in which the pains spread to the other leg originated in the shock of her sister's death. (See below.)

During the first period of treatment, after the above facts had been ascertained, she improved considerably. Variations in her condition could be traced to definite causes, such as the account of a friend's illness recalling to her mind her father's death, or the sight of a child reminding her of her dead sister's child. The author thought that the painful area on the thigh increased in size with the accession of fresh "psychical traumas." As after a time the patient made no further improvement, she was again put under the authors' "cathartic method" of treatment. It was then elicited that, as above mentioned, she had fallen in love with her brother-in-law. The news of her sister's illness had reached her when she was living at a considerable distance; and after a long and tedious journey in anxious suspense, she arrived only to learn that her sister was dead. She was horrified to find that the first thought that arose involuntarily to her mind, and against which her whole moral being revolted, was that her brother-in-law was now free to marry her. Then occurred the "conversion" of psychical into somatic innervations; this idea, so repugnant to her moral sense, being directed into somatic channels, and becoming the origin of fresh hysterical symptoms. The author then put clearly to his patient what he had gathered from her more or less rambling statements. This threw her into great agitation, and she denied what she had said. It was shown

her that no other interpretation could be placed on her own recitals; and subsequent interviews with her mother confirmed the accuracy of the main facts. The mother further said that no realisation was to be expected of the patient's wish to marry her brother-in-law. The subsequent history of the case was that after a period of illness she completely recovered her health.

Dr. Freud, commenting on the case, remarks that there was no evidence of hereditary tendency to nervous disease. The germs of hysteria were developed during the long period of strain and anxiety when the patient was nursing her father; his illness prevented her from paying attention to her own symptoms—"retentions hysteria"; had he recovered there would have occurred a rebound to a normal state of health; as he died, all the factors present necessarily led, after a short interval, to an outbreak of hysteria. The onset of pains in the thigh coincided with a conflict between her duty to her sick father and her erotic inclinations. The erotic ideas were forced out of consciousness, but replaced by physical pains. This conflict would probably take place many times over, and its consequences be thereby accentuated. Later, a conflict of the same kind arose between her love for her brother-in-law, both during her sister's life and after her death, and her moral feelings, followed by the development of fresh pains in the course of a period of exhaustion, the result of a fresh bout of nursing during her mother's long illness. Her love for her brother-in-law does not seem to have been consciously clearly realised, but remained as a "foreign body," apart from the other contents of consciousness, and not subject to associative interchange.

In their preliminary communication the authors were careful to speak of "the psychical origin of hysterical phenomena" rather than "of hysteria," because they do not wish to claim for their view an unlimited importance. They neither believe that all hysterical phenomena arise in the above described way, nor do they agree with Möbius that they are all dependent upon idea. They hold hysteria to be a form of disease discovered empirically from clinical observation, and the name a convenient clinical term like pulmonary phthisis. The knowledge thus empirically gained has been extended and simplified, but not destroyed, by the growth of knowledge. Though research has shown that pulmonary phthisis comprises different diseased processes, excited by the tubercle bacillus and by the micro-organisms of suppuration, &c., yet the term is rightly preserved as a clinical entity. So the term hysteria must remain; although

further knowledge may establish that its phenomena are due to different causes, some of psychical, others of non-psychical nature. Möbius's definition that "all those morbid symptoms are hysterical which arise from idea" is objectionable, because it breaks up hysteria as a clinical group of symptoms.

It seems certain that many hysterical phenomena do not arise through idea. Dr. Breuer takes as an instance hysterical pains; he admits that some of these do often arise directly from idea. But besides great vivacity of representation, abnormal sensitiveness of the pain-conducting and pain-receiving apparatus seems to be required. Though the expression, pain-hallucinations, vividly expresses the nature of these neuralgias, the ideas or memories concerned are never sufficiently intense to have that character of objective existence which marks an hallucination. In healthy persons memories of bodily pains cannot be called up with the same vividness as visual and auditory memories; in deep sleep pain is not dreamt of unless some real sensation of pain is present. He postulates, therefore, an abnormal irritability of the sensory centres for pain to explain the readiness with which "pain hallucinations" occur in the hysterical. In an arthralgia, arising from slight injury to a joint, a psychical element is present, but the affection can scarcely be said to originate through idea. So with pathological lowering of sensation. It is improbable that general or local analgesia and anæsthesia are produced through idea; and if Binet's and Janet's views are confirmed that hemianæsthesia is connected with a peculiar psychical state—*i.e.*, narrowing of consciousness—it is psychogenic, but not "ideogenic"; and, according to Möbius's definition, is not hysterical. Dr. Breuer would, therefore, say that many hysterical phenomena, probably more than we are at present aware of, originate through idea; but the fundamental morbid change is an abnormal irritability of the nervous system; and it is a further question as to how far this is of psychical origin.

Passing on to the question of what constitutes a "nervous" person, the author argues as follows:—Sleep and waking form the two extreme conditions of the nervous centres; sleep differs from waking in that in the former the association and conduction-paths in the brain for the excitation of the psychical elements (? cortical cells) are blocked. He further supposes with Exner that during waking there is a state of intra-cellular tension, that the conduction-paths are in a condition of tonic excitation, and that this intra-cerebral excitation is bound up with the power of conduction. In a man expectant and ready for some mental

or physical work, the tension or irritability of the conducting paths in the brain is increased. This involves wear and tear, and cannot be indefinitely maintained. Sleep means the restitution of brain energy, and unlike muscle, which only enters in action when stimulated, the brain begins to act when restitution is complete. If the brain remains at rest too long—no demand being made on it for mental or bodily work—a desire or necessity for activity is aroused, which, if not carried into effect, becomes painful. There is, therefore, postulated a *tendency to maintenance at a constant state of the intra-cerebral irritability*. If a proper means for its discharge be not found, and it is in excessive amount, the excess passes off in purposeless motor activities. The maintenance of the above-mentioned condition of strain or expectancy at appropriate times is essential to the performance of the best work; but if constantly recurring on inadequate or unsuitable occasions, it is hurtful; and is the characteristic state of the *nervous* individual. Certain organic sensations connected with the primary needs of the organism, *e.g.*, need of oxygen, are bound up with special mechanisms co-ordinated for their satisfaction, and are further dependent on chemical changes in the constitution of brain elements. The sexual functions, with their imperative need for satisfaction of the sexual sense, form a transition stage between the above elementary sensations and the psychical emotions. Emotion disturbs the dynamical equilibrium of the central nervous system, and causes an increased excitability. This increased excitability is discharged by means of the motor activities bound up with the emotion. Thus, in anger, the resulting wrathful actions lessen and relieve the increased cerebral activity. Emotions, such as terror or anguish, have, however, a depressing effect, and the disturbance they cause in the central nervous system only disappears through a gradual adjustment. If no such motor outlet is afforded to the excessive intra-cerebral irritability thus arising, the disturbance may still gradually disappear in healthy men; but in many persons abnormal reactions will occur, that is to say, there is an abnormal discharge of emotional movements.

Next there is a tendency in hysteria to change psychical innervations into physical (bodily movements). The motor activities by which relief is normally given to the increased intra-cerebral irritability produced by the emotions, are fixed and co-ordinated movements; but an excessive irritation may break through the usual paths and stream out in elementary movements; these are opisthotonus, representing the maximum effort of all the muscles,

and kicking and struggling. Such abnormal reactions to emotion are not confined to hysteria, and are only to be considered hysterical if they occur spontaneously without the incitement of an objectively grounded emotion.

Similarly with regard to the revival in memory of such past emotions. If at the time that an injury or insult is received, the recipient is able to freely express his indignation; subsequent recall of the injury in memory is attended with little excitement or irritation. Should, however, he have been unable to express his anger, recall of the event is associated in memory with the angry words, and forms the "psychical reflex" of the state of irritability. If the original emotion has been discharged by an abnormal motor action, the latter will also be recalled; in other words, the irritation arising from the recall of the emotion bound up with the conception of the injury is changed or converted into a physical innervation. Frequent repetition may so firmly establish this reflex act that the emotional element drops out; and, finally, as conceptions, unaccompanied by emotion, are far less vividly recalled in memory, the concept may also disappear, and the change into a motor act be perfect. The intra-cerebral irritation of the emotion has been diverted into a process of excitation of peripheral paths; and the concept, originally of emotional origin, recalls now, not the emotion but the abnormal reflex act. Further, the occurrence of the particular reflex act which may be present in any individual case, will often be determined by some emotion or voluntary action concomitant at the time of the original disturbance (path of least resistance).

Strong sensory impressions concomitant with the original emotion ("affecte") may accompany its revival in memory, not as a remembrance, but as an hallucination. In the higher grades of hysteria there often exists, between the original emotional disturbance and its (motor) reflex act, an active group of associated concepts which the author names "*Determinirung durch Symbolik*." In very many cases these hallucinations are incomprehensible, because the key to their origin is unknown to us. The disturbances (experience) which set free the original emotion whose irritation has become changed into physical innervation, the author calls a "psychical trauma," and thus assimilates cases of hysteria originating in this way to those of traumatic origin in the ordinary sense.

The psychical causes of pathological (hysterical) manifestations are religious doubts, conflict between evil inclinations and the moral sense, inability to follow in the path of duty, and the

sexual feelings and impulses which are especially potent. The pathological change may be in the disposition or temper (as in the conditions termed *Angstzustände*), or in the physical sphere, e.g., vomiting, nervous cough. Relief can be afforded by means of speech, to such states arising in this way. This has been long recognised in the Roman Church, by the institution of confession. Benefit may, however, be obtained from confession without a priest or absolution. To this whole group of cases, the authors give the name of "*Rétentions Hysterie*." There is another group of cases which Freud has described under the title of "*Abwehr-Neuro-Psychoses*." By "*Abwehr*" is meant the voluntary suppression of painful concepts, of concepts which a man feels to be injurious to his life's activities, or to his self-esteem. It is not easy to understand how a concept may thus be voluntarily forced out of consciousness, and cut off from its normal associations, but the corresponding positive process is sufficiently established, i.e., the concentration of the attention on a concept, and we know just as little how this is actually accomplished. Concepts separated from consciousness remain also cut off from the wear and tear of cerebral activity, and preserve their emotional accompaniments unaltered.

A third group of conscious states remains withdrawn from the wear and tear of thought, not voluntarily, but because they are forgotten, having originated in, and having been associated with emotions in states like hypnosis and the conditions allied to it, for which amnesia exists in the waking state. These are of great importance in the study of hysteria.

Conditions resembling hypnosis occur in the so-called hysterical hypnoid states. The authors describe the occurrence of states like hypnosis in hysteria, whose importance consists mainly in the amnesia present in them, and in their power to produce later that division or splitting up of the normal consciousness which is so important in severe hysteria. Yet they limit the importance of these states to the origination of somatic phenomena from idea, the transformation of psychical into physical innervation. Division of consciousness may also occur through voluntary amnesia in "*Abwehr-Hysterie*," by which concepts, or group of concepts, are shut off from the interchange of normal association.

These hypnoid states in hysteria alternate with waking states, and vary in frequency and duration; the dreamy condition to which they give rise often deserves the name of hysterical delirium. They differ from induced hypnosis only in their spontaneous origin and, as the name given implies, in being less well defined.

The mental contents of hypnoid states are shut off from those of the normal or waking life by amnesia, so that they cannot undergo correction, and, owing to loss of criticism and control by other percepts or concepts, may give rise to the most deranged and persistent hallucinations. A complicated irrational or "symbolical" nexus may arise between the pathological phenomena and their exciting cause, often dependent on ludicrous sound-similarities and word-associations. Hysterical symptoms may be restricted to the hypnoid condition, so long as this alternates with the waking state, and be intensified by repetition. The mental image, or idea, which is the cause, of the condition, never emerges clearly into consciousness. Change of psychical into somatic symptoms may take place during these hypnoid states.

In one case the origin of these states arose out of fits of reverie, which were the result of long-protracted anxiety. The author thinks they often arise in brooding over love disappointments, and during long hours of nursing sick relatives. It is possible that such states may arise spontaneously in those hysterically disposed without the co-operation of the emotions. It might be thought that an intelligent patient, capable of self-observation, would recognise the connection between idea and hysterical phenomena. The inner causal nexus would be obscure, but the outward connection would be recognised. Patients, however, as a rule, deem themselves to suffer from independent corporeal phenomena. The authors believe that their observations show that the *psychical trauma* remains in memory for a long time as an active working (irritative) agent, outside the patient's consciousness, and comparable to a foreign body. A division of consciousness or of the psychical activity results, and is of fundamental importance in the elucidation of complicated hysterical phenomena.

Sub-conscious Perceptions — Division of Consciousness.—The existence of sub-conscious mental states, or perceptions is undoubted. Understanding by "sub-conscious" states those in the background of consciousness, they make up a large part of the psychical accompaniments of normal brain action. The ability of perceptions to affect consciousness is largely dependent on the extent to which they are associated with emotion. If they at once set free a motor act, by passing at once into motor channels they lose their clearness and intensity as perceptions. The following example is given of the mode in which such perceptions may give rise to hysterical symptoms. A man whilst eating sustains some intense emotion to which there is no corresponding

reaction (*e.g.*, by speech, action or weeping, &c.) Subsequently each attempt to eat excites loathing and vomiting, and relief is not obtained from these symptoms until an outlet, by speech or some other means, has been afforded to the pent-up emotion. The patient is ignorant of this connection between cause and symptoms, which may only be discovered with difficulty by aid of hypnotism.

In such individuals presentative or representative states may be conscious or sub-conscious; they may or may not be capable of affecting clearly defined consciousness; thus is constituted a division of the psyche, which, in hysteria, is divided into two relatively independent parts. The authors do not say that all hysterical phenomena arise in this way, but rather that "that division of the psychical activity, which is so striking a feature in the known cases of double consciousness, exists in rudimentary fashion in every marked case of hysteria, and that the tendency for this dissociation to occur is the fundamental phenomenon of the neurosis." Professor Janet regards this division of psychical activity (personality) as dependent on innate psychical feebleness. In the normal man it is only when the attention is concentrated on one subject, or when the image or idea is exceptionally powerful, that it occupies the whole field of psychical activity, otherwise it arouses, by association, a number of others, which support or inhibit it. In hysteria, however, each one of any degree engrosses the whole psychical activity, and is thus shut off from associative interchange. The author does not agree with this view, that there is innate feebleness of mind, which he thinks is negated by fact that the most highly gifted persons may be hysterical, but considers that the due action of the mental powers may be interfered with, or enfeebled, by the co-existence of two heterogeneous groups of perceptions or ideas, causing loss of power by diversion.

The co-existence of day dreaming, or of frequent strong emotional states, with normal psychical activity, tends to a pathological division of the psyche. If the two co-existing groups of conceptions no longer have a similar content, if one group is sub-conscious the division is accomplished; hence arise hypnoid states.

How far does the lately-acquired knowledge of division of consciousness aid in the understanding of hysteria? First, many apparently physical phenomena can be traced to psychical causes of which the patient is not fully aware. The sub-conscious representations, though never, or very rarely, entering into the waking thoughts, yet influence them, and also influence the processes of

association. Thus they determine the disposition, and render comprehensible the "tempers," and motiveless and inexplicable "humours" of hysterical patients. The emotions are reinforced by the sub-conscious representations, and, from the oscillation between primary and secondary states, the psychical powers are weakened, and the patients seem devoid of energy, childish, or even imbecile. In hemianæsthesia the loss of impressions from one half of the body tends in the same direction. Further, the increased susceptibility to suggestion is explained by the poverty and incompleteness of the sub-conscious separated part of the psyche, and the disturbance of association.

With regard to the original disposition in, or mode of development of hysteria, the author, as above stated, rejects Janet's view of innate psychical weakness, and substitutes an abnormal irritability of the Central Nervous System, through which the sensory, vasomotor, and visceral apparatus are abnormally accessible. Thus arises hyperalgesia of a part to which attention has been directed by some slight lesion; *e.g.*, hysterical joints, palpitation, nervous asthma, &c. The psychical origin of hysterical anæsthesias, analgesias, and contraction of visual fields is, he thinks, not yet established. The stigmata, the especial hysterical symptoms which form the diagnostic basis of hysteria, arise out of the fundamental disposition, and do not originate in idea. Next to them come those phenomena which are "ideogenous," and are simply the conversion of emotional excitations into somatic (physical) symptoms. The author lays stress on the fact that the most frequent and most powerful emotions to suffer this "conversion" are the sexual ones; that the sexual factor is by far the most potent and most pathologically fruitful in the production of hysterical phenomena. The observation of the ancients expressed in the word hysteria is nearer to the truth than the new views which put sexual disturbance almost in the last line. Next to the sexual factor the most important is terror—the essentially traumatic hysteria. The hypnoid states, or tendency to auto-hypnosis, form the third constituent of the hysterical disposition. The origin of these states is doubtful, and how sexual influences are concerned is not known. The mode of origin of division of consciousness in hypnoid states has been alluded to, and these states are responsible for the origin of hallucinations and of motor acts independent of the conscious will.

Psychotherapy of Hysteria.—The method of treatment followed has been described in BRAIN (*loc. cit.*); briefly it consists in getting the hypnotised patient to fully express in words the mode

of origin of his symptoms with the accompanying circumstances of the onset. Dr. Freud names it the *cathartic* method of treatment, and remarks that two difficulties had to be met at the outset: first, that all patients could not be hypnotised; and second, that a diagnosis could not be based on the results of treatment. For all hysterical disorders are not curable by this means, and some other neuroses are. He, therefore, resolved to investigate and treat all neurotic patients in the same way, and to allow the diagnosis of hysteria to depend on the result of the enquiry. As a result of this investigation he was forced to the conviction of the paramount importance of sexual disorders in the ætiology of the neuroses; and further than this, that different forms of sexual disorder produce different forms of neurosis. He differentiates the following forms of neurotic disorder:—

(1) Neurasthenia, presenting constant clinical features, in which a psychical mechanism plays no part.

(2) “Zwangsneurosis,” characterised by uncontrollable and frequently-returning thoughts and ideas, and having a similar etiology to hysteria. Imperative ideas.

(3) “Angstneurosis,” split off from neurasthenia, and characterised by anxious forebodings, morbid fears, and hyper-æsthesia to pain. It is not of psychical origin, but influences psychical activity, arising through repeated physical overstrain, itself, again, of sexual origin. It is to be distinguished from hypochondriasis, in which there is a constant dread of bodily illness.

(4) Hysteria.

The above neuroses exist frequently in combination in the same person. Neurasthenia and the “Angstneurosis” are most frequent in the pure form. It is scarcely possible to consider hysteria apart from sexual neuroses. He thinks hysteria may in every respect be treated of independently from other neuroses, except from the point of view of therapeutics. From his own point of view it is most important to separate clearly the part played by hysteria in the formation of a mixed neurosis, for the cathartic method of treatment is effectual in hysteria, is of no avail in neurasthenia, and only to a limited extent in the “Angstneurosis.” He does not claim to remove all hysterical symptoms, but attributes failures to difficulties in dealing with individual cases rather than to faults in the principle of the method. The cathartic method treats symptoms and not causes. It is most effectual in dealing with symptoms remaining after an acute hysterical paroxysm; here it meets all indications, and gives

prompt and enduring results. In acute hysteria, *i.e.*, period of active production of hysterical phenomena, it is of less value; because the symptoms, if removed, soon recur. In monosymptomatic hysteria and in chronic cases, with continuous production of hysterical symptoms, the cathartic method is especially valuable. Dr. Freud further insists on the necessity for the fullest confidence between doctor and patient, if success is to be attained; that in his hands hypnosis has led to no bad results; and that his method of treatment may often be combined with the Weir-Mitchell treatment, with most advantageous results, which could never be obtained from the latter alone. The author, at the outset, was confronted with the difficulty that all patients are not hypnotisable. He found that in many of these cases, by repeated close questioning, some information could be obtained of the primary origin of the symptoms. To get information in this way required great mental exertion, in order, as he supposes, to overcome a psychical resistance in the patients, tending to prevent the emergence of these pathogenic ideas into consciousness. He notes that all these pathogenic ideas have the common attribute of painfulness; they were attended with emotions of shame, remorse, or injury. In the evolution of the neurosis this painful impression had been "converted," forced out of consciousness, but had left traces of its presence. The same power which had originally forced out of the conscious life the painful impression now resisted his efforts to revive it in memory. Hence, in hysteria, the not knowing implies disinclination to know; and the object of treatment should lie in overcoming the resistance to normal processes of association. As a result of these investigations, the author was led to adopt the following method of procedure, to which he attaches no specific importance other than convenience. The patient lies down with his eyes closed, and the observer, exercising light pressure with his hand upon the patient's forehead, tells him that during this pressure a picture (visual memory) will rise before his eyes, or a remembrance of some past event will be recalled, which he must describe at once without reserve, and as fully as possible, of whatever nature it may be, even if it appears to him to be disagreeable, trivial, or not to the purpose. In this way a knowledge of the pathogenic concept is obtained, occasionally directly, but more often after a long chain of associations has been started, which finally leads to the desired result.

Perseverance is necessary to success, the method may fail the

first time and afterwards be completely successful. It is essential that the patients should simply report what they visualize, or otherwise recall, and not their own reflections. Visual memories are most easy to deal with. The vision fades from memory, after its description by the patient, unless there still remains something of importance to be elicited in connection with it, in which case it stays obstinately before him.

As a rule the method is a trustworthy one, and succeeds in every case; it must be combined with close observation of the patient's expression; one soon learns to distinguish the expression of mental rest, which follows on a successful employment of the method, from the state of strain and emotion existing previously. The full results to be gained by the procedure are only reaped at the cost of much time and patience. After a time the contrast between the results at the first and those attained at later séances are very striking. The patient can be intellectually interested in the process, and thereby further advance made. If the question were asked whether recourse to hypnotism would not render the carrying out of the "cathartic" method more thorough and easy, the author, so far as his experience goes, would reply in the negative.

In a former paper the authors made the classification of hysteria into

1. "*Abwehr Hysterie*"—acquired hysteria, in which an unbearably painful concept is forcibly expelled out of consciousness and transformed into some somatic disturbance.

2. "*Hypnoid Hysterie*," in which there is a division of consciousness, one group of concepts being completely separated, or shut off, from the ordinary consciousness.

3. "*Retentions Hysterie*." The authors are not now prepared to recognise the second form as distinct in origin from the first, and do not finally pronounce upon the third form.

They go very fully into the method of carrying out the above mode of treatment, and the difficulties likely to be met with; the analysis of the symptoms, and of the psychological changes on which they depend, proceeding step by step until a full acquaintance with the mental state of the patient, and of the conditions which originally gave rise to the neurosis and of the subsequent stages in its development, has been gained.

I have in the above endeavoured to give as concisely as possible the authors' views. The most important points in them seem to be that they afford an explanation for the origin of some of the somatic symptoms of hysteria in psychological causes. They still

leave unanswered, however, the mode of origin of the fixed "stigmata," such as hemianæsthesia, contraction of visual fields, &c. Perhaps the strongest arguments in their favour are, first, the approximation afforded on their views of ordinary cases of hysteria to cases of traumatic hysteria, in which the cause, the way in which it acts in producing hysterical symptoms, and the nature of the symptoms so produced, can be most easily followed out and studied; and, secondly, that due importance is attached to the undoubtedly great part played by the emotions in the evolution of hysterical symptoms, and some explanation given of the way in which they do this. It may further be added that their theories give, in psychological terms, an explanation of some of the characteristics of hysteria, long established by medical observation, and some of them matters of common knowledge. It is interesting to note a return, in part at least, to the old theory of the origin of hysteria in sexual disorders, especially as the tendency of late years has been to attach very much less importance to them. The authors adopt the theory of a division of consciousness as accounting for many hysterical symptoms and states; in the ordinary run of hysterical cases, this, if present, must be so in a very rudimentary form. They also pre-suppose, as underlying hysterical phenomena, an irritable state of the nervous system. It is in what exactly constitutes this "irritable state," and how it arises, or, if we prefer so to call it, the "innate mental feebleness" of Janet, that the key to the problem of hysteria is to be found; and it is just as to this point that our knowledge is most severely limited.

The book is written in an interesting style, and bears the stamp of originality, and of much patient and painstaking observation. Into the question of the advisability of penetrating so intimately into the most private thoughts and concerns of a patient I do not enter; it would seem likely that the patients would, in many cases at least, strongly resent it. The necessity of bearing in mind, in studying hysterical patients, the great readiness with which they respond to suggestions, may be reiterated, as the weak point in the method of investigation may perhaps be found here. The danger being that in such confessions the patients would be liable to make statements in accordance with the slightest suggestion given to them, it might be quite unconsciously given to them, by the investigator.

J. MICHELL CLARKE.

Traité clinique et Thérapeutique de l'Hystérie d'après l'Enseignement de la Salpêtrière. Par le Dr. GILLES DE LA TOURETTE, Professeur Agrégé à la Faculté de Médecine de Paris, Médecin des Hopitaux, &c. Preface de M. le Pr. J.-M. CHARCOT. Seconde Partie. Hystérie Paroxystique avec 63 figures dans le texte et un portrait à l'eau-forte du Professeur Charcot. Vols. I. and II. pp. 1146. Paris: E. Plon, Nourrit et Cie, 1895.

THESE two volumes form the second and third of M. Gilles de la Tourette's work, and bring it to a close. They deal with the paroxysmal manifestations of hysteria, defined as the episodes or incidents occurring on the general basis afforded by the presence of hysterical stigmata, and characterised by suddenness of onset and of disappearance. The attack may be acute or prolonged, the latter forming the *état de mal*, or, in the sphere of mind, may constitute a permanent "second state." It may be said at once that the book is a very complete one, and that every kind and degree of hysterical manifestation is here treated at length. In this notice the author's views, which embody those held at the *altpêtrière*, will be given on those features of hysteria which are of most interest and importance, or about which there is considerable divergence of opinion. The first volume, of which a digest will be found in BRAIN, 1892, pp. 522 *et seq.*, dealt with "normal" hysteria, *i.e.*, the ordinary state of hysterical persons between the paroxysms; the so-called "stigmata," sensory and motor, being taken as the evidence of the presence of the hysterical state.

In treating of hysterical fits Charcot's description of the paroxysm in hysteria major is adhered to, and his well-known table of phenomena, and P. Richer's beautiful drawings, are reproduced. At the same time the author admits that the fully-developed typical attack as thus described is rare; he also admits the part which suggestion may play on other hysterical patients in the same ward, with a sufferer from these fits, but thinks that the description should be retained to aid in the elucidation of the incomplete fits. He combats the view that has been brought forward by Bernheim and others, that these fits are a growth of the *Salpêtrière*, and peculiar to it, by quoting published cases of other authors, notably Andrée and Knoblauch's account of the occurrence in German soldiers of fits corresponding exactly to

Charcot's description. It must be admitted, we think, that attacks in the complete form are very rarely observed, and are a matter rather of theoretical, than practical importance. The author gives a long and vivid description of a patient in one of these severe attacks. A short account of the ordinary, milder, hysterical fit, such as is commonly met with, is desirable. Whether it is true that the milder attacks represent incompletely the more severe ones, some phases of the latter only being present, must be difficult to prove satisfactorily. Certainly in the immense majority of hysterical patients only the minor attacks occur, and only very distantly resemble the severest form. The greatest point of resemblance lies in the frequent occurrence of a state resembling the somnambulistic or hypnotic condition—the third period in Charcot's scheme—in which the patients react readily to suggestion from without.

Amongst minor points the author says that convulsive attacks, though less frequent in men, when present are severe, and that the period of delirium is the most marked feature of the fits in children, and may be the only one. He divides the aura into (1) prodromal phenomena, and (2) aura proper. The most interesting and varied of the prodromal phenomena are in the intellectual or psychical sphere, and have generally a sombre character. He gives elaborate researches, which involved much labour and were carried out with due precautions to avoid error, on the nutritive changes during a convulsive attack, as shown by the urine. All the urinary excreta are diminished relatively to the normal during an hysterical, whilst they are augmented during an epileptic fit. The urine examined for twenty-four hours from the onset of an hysterical fit shows a diminution in the amount of urea and total solids, whilst the ratio of the earthy to the alkaline phosphates is changed, the earthy being passed in increased ratio; the normal proportion being 1 to 3, in hysteria the amounts passed of each may be equal. In the cases investigated, variations in amount of food were carefully excluded. The whole formula should, however, be taken into consideration, and not merely the inversion of the ratio of the phosphates. He replies, at considerable length, to those who have combated the accuracy of this statement; and the conclusions arrived at appear to be justified by the data given. Considerable stress is laid on this as a method of diagnosis between hysteria and epilepsy.

Do hysterical fits ever terminate fatally? This subject is discussed in an interesting section, in which the historical cases

of death from hysterical seizures are reviewed. It comes out that in nearly all the cases the accuracy of the diagnosis is open to question; and the author concludes that a fatal termination in the course of an hysterical fit, whether simple or prolonged, is of the rarest occurrence; and that when it does occur, attacks of laryngeal spasm most frequently cause it. Finally, for a fatal result to take place, the co-existence of organic disease seems to be necessary, the fit being merely an adjuvant cause. Hysterical fits may be very rapidly repeated at short intervals, during which the patient suffers from the effects of the paroxysms (*état de mal*). One characteristic of these attacks is the rapid loss of weight that occurs, the loss being made up almost as rapidly when the attack is over. The urine shows the characters given above, and the quantity passed is of some prognostic importance, as at the onset there is a fall in the amount, which remains stationary during the attack at a lower level than health, and towards its termination begins to rise again to the normal. This general rule is, however, vitiated by exceptions, due to the frequency of relapses. The chapter on lethargy, or hysterical sleep, or trance, is particularly valuable. It begins with an account of the history of the subject; for in all ages this peculiar condition has excited great interest, and in many of the most ancient cases the diagnosis is quite clear. With regard to diagnosis, the doctor may be called to see a patient apparently comatose, but really in hysterical sleep. In women a history of hysterical attacks can generally be obtained, and other hysterical symptoms observed; but in men such a condition may be the first manifestation of the neurosis. The onset may be sudden or gradual, and is generally preceded by prodromal symptoms. Frequently active hysterogenic zones are to be found, together with general anæsthesia. Even if there is general muscular relaxation, trismus, or rapid intermittent contractions of the eyelids are present. The author recognises attacks which last only some seconds or minutes! They may certainly last for weeks, but observations of a duration of months or years he considers doubtful. The patients are not necessarily fully unconscious; instances are on record where they have heard and appreciated the preparations for their burial without being able to give any sign. The attack terminates suddenly by a convulsion or an outbreak of tears or laughter. Death never seems to occur; the one case on record being of doubtful nature. Similar changes in the urine to those above described as occurring during the fits are present, and are independent of the amount of food taken.

Under the name of katatonia, Kahlbaum described what he believed to be a special disease. In this a stage of melancholic depression precedes a stage of excitement, which is followed by a catatonic stage, presenting the characters of melancholia with stupor. In the latter motor phenomena are present, which bear a close resemblance to hysterical sleep, with cataleptiform contractions. The patient remains in a lying posture, with abolition of general sensibility and of the special senses, vibratory tremor of the eyelids, trismus and cataleptiform contractions of the limbs. This condition (catatonia) seems further to be sometimes associated with hysteria. The former, however, is distinguished by a history of antecedent mental trouble, whilst the onset of hysterical sleep is generally abrupt, and, unlike catatonia, never ends in dementia or death. M. Gilles de la Tourette does not admit that there is any well authenticated case in which hysterical sleep has lasted more than from five to six weeks consecutively, whereas the catatonic condition has been known to last for eleven years.

Catalepsy itself he does not consider, so far as its objective phenomena are concerned, to belong to hysteria; it is to be regarded as a group of symptoms, which may appear under various conditions, *e.g.*, in hysteria, in which case it frequently accompanies the fits, or in mania and melancholia. When it succeeds hysterical fits the different cataleptic attitudes are assumed under the influence of hallucinations. In hysteria catalepsy may be merely mixed up with other phenomena of the attack, or may be the dominant and apparently primary symptom. The former is by far the most frequent. Prolonged cataleptic conditions in hysteria are to be regarded as attacks of prolonged hysteria. Here, again, both in diagnosis and prognosis the author largely relies on the characters of the urine.

With regard to hypnotism, the author urges the view held at the Saltpêtrière, that hysterical persons only are really hypnotisable. General experience has, however, shown that the phenomena of hypnotism are not confined to the hysterical. Somnambulism he regards as merely a manifestation of hysteria, and regards the "second states" of double consciousness as a prolonged hysterical somnambulism, in which the convulsive phenomena play only a minor part.

In this connection the curious affection, called "ambulatory automatism," is mentioned. Cases are met with in which the patients wander off on some vague but uncontrollable impulse,

leaving their homes and avocations, and after a longer or shorter time, return to their normal state of mind, with no memory of anything that has occurred in the interval. If the attacks occur often they lead a kind of dual existence. This condition has long been known to occur, in greater or less degree, after epileptic fits. Charcot and Pitres first described it in hysteria. According to them, the hysterical differs from the epileptic variety, chiefly in not being amenable to bromides, and in the fact that, if the patient is hypnotised, in the hypnotic state he remembers all that passed during the period of "automatism." Unfortunately, no investigation of a patient whilst actually in this condition has yet been made. There is no doubt of the affinity of these states to somnambulism; and the author further regards them as equivalent to the third and fourth period of the hysterical fit. The view which regards hysterical somnambulism and second states, as modified periods of the fits, seems to rest chiefly on similarity, and is, perhaps, a point which requires further proof.

The views as to the nature of hysteria, held by M. Janet and his fellow-workers (BRAIN, vol. xvii., p. 129) do not commend themselves to the author, and he dismisses them with a brief reference. He thinks that they do not account for the physical phenomena of hysteria, and that in place of accurate clinical observation, these writers have merely applied to hysteria preconceived psychological theories. Some of their experiments have, however, thrown remarkable light on the true nature of hysterical anæsthesia, &c. There is a very full account of cutaneous trophic disturbances; the author states he has been careful to accept only facts of undoubted authenticity. Vasomotor disturbances are so common as to form a special feature of the neurosis. A perusal of the cases of the various skin eruptions met with in hysteria, suggests that great care and discrimination is needed before directly attributing them to the neurosis.

The occurrence of spontaneous ecchymoses, or hæmorrhages, from the skin and viscera, is so interesting that I give a little longer notice of M. G. de la Tourette's account of this subject. One of the most remarkable and best authenticated cases of hæmorrhages from the skin is that of Louise Lateau—*la stigmatisée Belge*—studied by Bourneville. In her case the bleeding points corresponded to the stigmata of crucifixion, and bore a remarkable resemblance to the celebrated bleeding stigmata of St. Francis of Assisi. This rare phenomenon is not confined to hysteria, but has been most frequently observed in it; at least one case in man is on record; and, in women, although it is often related

to the suppression or appearance of the menstrual periods, it has no constant connection with them. In many patients the bleeding seems to be due to the secretion of a blood-stained perspiration, rather than an actual hæmorrhage; but in others the appearance was that of ordinary blood. The bleeding has most often taken place from an erythematous or vesicular patch, and sometimes from an indolent ulcer. In one case the hair follicles were the seat of hæmorrhage, which was confined to the hairy parts of the body, little drops of blood forming around the roots of the hairs; and cases are recorded when the bleeding took place from the ceruminous glands of the external auditory meatus. The cutaneous hæmorrhage is often accompanied by epistaxis, hæmatemesis, or melæna. Although the quantity of blood lost has been considerable in a few cases, no danger to life seems to have ever occurred. Pains, or sensations of burning, or an hysterical fit, sometimes precede the hæmorrhage. The reader will find ample information on this subject in the book. Hypnotism has, perhaps, thrown some light on this obscure subject, by showing that cutaneous lesions may be the result of suggestion. For instance, a postage stamp was stuck on a patient's shoulder in a spot that he could not reach; he was hypnotised, and told that the stamp was a blister. He was placed under close surveillance during the rest of the day, and sent to sleep for the night; in the morning a blister was found. Again, blisters were placed on the chest of a tuberculous patient, and on both arms of a hysterical patient; the latter was told during hypnosis that the plaster on the right arm would not raise a blister. Care was taken to watch this patient closely; and it was found that blisters duly appeared on the first patient's chest, and on the second patient's left arm, but not on the right. An *hæmoptysis* due to hysteria is now described. It occurs in men as well as in women, which negatives the view that these hæmorrhages are vicarious of menstruation. The author thinks its more frequent occurrence at the menstrual periods due to the fact that convulsive attacks, which provoke hæmorrhage and other hysterical vasomotor phenomena, are so common at these times. Hysterical hæmoptysis may occur during the fits, or in the intervals. Such patients often suffer from painful points or areas in the chest wall, and from an hysterical cough. In most patients the physical signs are much less than would be expected from the abundance of the hæmoptysis, intensity of cough, and aspect of the patient. Local signs, such as crepitation, may come and go from day to

day. The author thinks that pulmonary congestion of hysterical origin exists, characterised by appreciable signs on percussion and auscultation. As a rule, which is not invariable, fever is absent; and in most cases the hæmorrhage is stated to be more abundant than in pulmonary tuberculosis. The author points out the great difficulties in diagnosis from the latter. He notes the frequency of phthisis in the hysterical; and thinks that there is no proof of the view that has been urged by some writers that hysteria is truly antagonistic to, prevents the development, and retards the course of phthisis.

Hysterical hæmatemesis has been observed under the following conditions:—An attack of acute pain and swelling in the epigastrium, the pain increasing and radiating into the back, comes on with the ordinary symptoms of the hysterical aura, *i.e.*, palpitation, præcordial anxiety, sensation of constriction of the œsophagus, giddiness, noises in the ears, and beating at the temples; this is followed by vomiting of blood, once or twice repeated; the patient passes then into a hysterical, lethargic state, which is not due to syncope from loss of blood, because it occurs when the loss of blood has been very slight. The blood may be pure or mixed with fluid from the profuse salivation, which often accompanies the attack. If these hæmorrhages are large or frequently repeated, as they sometimes are, for five or six days, the patient falls into a profoundly anæmic state. In the course of some investigations into the diagnosis between hysterical hæmatemesis and that due to simple round ulcer of stomach, the author found that of eighteen patients (thirteen women, five men) suffering from well-marked symptoms of gastric ulcer, six were hysterical. He suggests that hysteria may be a determining factor in the etiology of round ulcer; and, reasoning from the analogy of the above-described trophic cutaneous lesions in hysteria, supposes that an ecchymosis (hysterical) of the mucous membrane may, under the action of the gastric juice, be converted into an ulcer.

Hitherto it has been generally held that, in a doubtful case of severe gastric pain, the occurrence of well marked hæmatemesis would decide in favour of a diagnosis of ulcer. The hyperæsthesia over the epigastrium, and excessive epigastric reflex, so commonly present in gastric ulcer, can hardly be adduced as evidence of hysteria. Hysterical girls are very often anæmic; gastric ulcer occurs most frequently in the anæmic; and one would expect, therefore, that gastric ulcer should be sometimes associated with hysteria. I think that clinical experience in this country, at

least, does not point to any closer relationship of the two affections; cases of gastric ulcer do not, as a rule, present evidence of hysteria. In the case of both hæmatemesis and hæmoptysis, an hysterical origin for it could not be accepted as satisfactory without the positive proof furnished by *post-mortem* examination; seeing that both gastric ulcer and pulmonary tuberculosis may be present without being recognised clinically. Healed tubercular lesions are found in the lungs *post-mortem*, when nothing in the patient's history pointed to a previous tubercular affection; and ulcers, healed or not, are also occasionally found in the stomach, whose existence had been unsuspected during life. In view of these facts it is hardly necessary to add that a diagnosis of hysterical hæmoptysis, or hæmatemesis, must be an uncertain one; and, if made, could only be acted upon with the greatest caution.

There seems no doubt that muscular atrophy may occur in hysteria. Most cases have been in men. Generally it is a complication of paralysis or contracture, and other trophic disorders are often present. The onset is fairly rapid; and it most commonly affects the whole limb, but sometimes only a part of it, such as the small muscles of the hand. The atrophy is accompanied by fibrillary tremor. The electrical change is a diminished reaction to both forms of current. Aid in diagnosis is given by the atrophy following on paralysis or contracture, with disturbances of sensation and other hysterical manifestations. There is a good account of hysterical fever, to which the reader may be referred, as this subject was treated fully in BRAIN, 1894, p. 196.

The section of hysterical paralyses and contractures is very full and complete, and it opens with an interesting historical introduction. Full justice is done to the work of Todd and other English writers on the subject. With regard to hysterical hemiplegia, the old diagnostic point as to absence of affection of the face, though generally, seems not to be universally true, many observations being now on record of paralysis of the lower part of the face. It is, however, rare and as a rule, slight, it varies from day to day, and individual movements may be dissociated and paralysed. It is often accompanied by spasm of the muscles of the opposite side of the face.

Labio-glossal spasm is more common than facial paralysis in hysteria; in the great majority of cases it accompanies hemiplegia, generally on the same side, but sometimes on the opposite. The affected muscles are affected with a rapid clonic tremor. The spasm has a tendency to spread to the neck muscles, especi-

ally to the platysma myoides. Trismus, common in the hysterical fit, is rare as a persistent affection, and is then generally accompanied by spasmodic contraction of other muscles. This labio-glossal spasm is little marked during repose, but comes out on movement. There is slight derangement of speech and deglutition. The excessive deviation of the tongue is a diagnostic feature; when the patient is told to protrude the tongue, it is bent round like a hook, so that the tip bulges out the cheek. Aid in diagnosis is also given by the presence of anæsthesia over those parts of the face and tongue affected by muscular spasm. Like spasm, hysterical facial paralysis is most obvious on movement, and very little marked during rest. There is no change in the electrical reactions in either spasm or paralysis. Both are generally transitory, but one case lasted three years, and showed no tendency to cure.

Hysterical hemiplegia, monoplegia, and paraplegia are fully discussed. With regard to monoplegia, it is said that the prognosis is variable, but bad, in male adults. Monoplegias, generally, are apt to persist (see BRAIN, vol. xvii., p. 294), and to be attended with trophic troubles. The extent of accompanying anæsthesia varies; so long as any anæsthesia is present, the paralysis is apt to return. In two cases of paraplegia of spastic type, in which the diagnosis of hysteria was not doubtful, incontinence of urine was present. Two cases of bed-sores occurring in the same affection are mentioned. In the differential diagnosis of hysterical from organic hemiplegia, stress is laid on the frequency of sensory disorders in the former; on the general absence of affection of the face, whilst if the facial muscles are affected, spasm, and not paralysis, is generally present; and on the fact that the tendon-reflexes are normal.

The author believes in a mode of action of hysteria, which he terms "auto-suggestions organiques." An example will best explain what is meant by this term. MM. Brissaud and Lamy report a case of brachial monoplegia, of historical origin, in a patient suffering from lead-poisoning; the supinator longus being unaffected, just as in ordinary lead palsy. They quote a similar case described by M. Potain, and conclude: "Hysteria has smitten the parts affected by lead poisoning." Similarly, the author says, that he has often observed hysterical hemiplegia and brachial monoplegia in syphilitic patients, who had been more or less affected by cerebral syphilis. Instead of the hemiplegia or monoplegia of organic origin, which had been cured by appropriate treatment, hysterical hemi- or monoplegia had

appeared, for whose provocation and localisation the organic lesion was responsible. The frequency with which hysteria complicates organic nervous disease is now generally accepted; and, *a priori*, one might expect that those parts previously paralysed from organic lesion would be more likely than others to be affected in a subsequent outbreak of hysteria. But that the hysterical should occupy the exact field of the organic paralysis to the extent described above seems to require further proof. In a case like the one of lead-poisoning, such a distribution of the paralysis would make the diagnosis of its hysterical nature one of extreme difficulty and of some doubtfulness.

There are good descriptions of hysterical paraplegia, pseudotabes, and of the various forms of contraction affecting the upper and lower limbs, especially of the hand and foot; this section on contractions having a number of excellent illustrations. The author brings forwards evidence and quotes authorities in favour of the view that many cases of tetany, and especially those cases of so-called essential tetany, in which no cause can be traced, are of hysterical nature. He thinks that a large part of the essential contractions of the extremities or tetany, in both mild and severe forms, are due to hysteria, and that hysterical tetany may show itself in epidemic form in children.

Following Charcot, three forms of astasia-abasia are distinguished. In this affection movements of standing and walking are alone affected; rarely is there any affection of sensation or of the muscular sense; and the reflexes and muscular nutrition are unimpaired. (1) Paralytic or paretic, in which there is weakness or difficulty in walking, or the gait is stiff and constrained. (2) Choreiform (type of flexion, Charcot), in which there is sudden bending or giving way of the legs, with consequent violent compensatory muscular contractions to prevent falling. (3) "Trépidante," in which walking is rendered difficult by contradictory movements, which stiffen the lower limbs and produce a sort of tapping, stamping, or clonic spasm. This latter, in exaggerated form, gives the saltatory variety long described as saltatory spasm. The paralytic form may give rise to difficulty in diagnosis from cerebellar tumour, especially as there may be marked vertigo. The pathogenesis of the affection is referred to a disorder of the complex co-ordinated spinal mechanisms for standing and walking.

The author gives a learned and exhaustive chapter on the various forms of cough, and of simulation of cries of animals in

hysteria. Some of the accounts of epidemics of this nature in former times are very curious. In one instance the nuns in a convent took to miauling like cats; the noise speedily ceased when the magistrate stationed a company of soldiers at the door, with orders to enter and chastise the miaulers!

Yawning, sneezing, and laughing are known to occur in paroxysms in the hysterical. One patient is said to have yawned eight times a minute, or 7,200 times in fifteen hours. These paroxysms may end in true convulsive seizures.

In hysterical laryngeal affections it is often very difficult to say whether we have to deal with a paralysis of the abductors, or spasm of the adductors; as in the case of the ocular and facial muscles, paralysis of one set is apt to be accompanied by spasm of their antagonists. A form of dyspnœa is met with in the hysterical, which, if its existence is not known, may lead to errors in diagnosis. The name, tachypnœa, proposed by Charcot, is more suitable than dyspnœa. The respirations may be 170-180 to the minute; they are shallow, and not noisy; the auscultatory signs are normal; there is no cyanosis, and the pulse is not quickened; the patient betrays no anxiety. The attacks last from three to four hours; are almost always preceded by symptoms like those of the aura of an hysterical fit, and often end in an outburst of tears.

In hysterical gastric affections, vomiting is considered to be the predominant symptom, and attributed to hyperæsthesia and spasmodic contraction of the stomach. The vomiting is constant, the food being rejected without effort or pain as soon as swallowed. All the food is not vomited, but the amount retained is not sufficient to prevent emaciation. The event is favourable, but there is a great tendency to relapse. The author attributes both hysterical vomiting and gastralgia to the presence of hysterical stigmata in the digestive tract. In gastralgia he supposes that there is an hyperæsthesia of the gastric mucous membrane; and from this point of view he recognises two clinical varieties: (1) cases in which the stomach is the seat of an hyperæsthetic and hysterogenic zone, and the skin over it is also the seat of anæsthesia or hyperæsthesia; the above zones are only rendered active as the sequel of a convulsive attack, or of irritation of an ovarian zone. (2) Cases less common, in which the hyperæsthetic gastric zone acts primarily. Food then excites an attack; and if not at once expelled, the symptoms increase in severity until they may even end in actual convulsions; pain being, however, the most striking symptom throughout. After such an attack is over, the

stomach becomes tolerant, and food is retained and digested. Marked changes in the composition of the gastric juice are not found.

In hysterical anorexia the psychical element predominates, though anæsthesia of the stomach may aid in producing the symptoms. The two psychical factors are the loss of sensation of hunger and of all wish or desire to eat. In the earlier stages of the affection, the patients display an unusual and morbid activity; later, they fall into a semi-comatose state, in which the only will-power shown is the refusal to take food. The fact that the stomach may one day not tolerate a drop of water, and on the next be able to manage a full meal, shows that it is not primarily affected, and that the disorder is a mental one. Relapses seem not to occur; a point in which this affection differs from similar conditions in the insane, and also from other hysterical disorders. The urinary excreta, liquid and solid, vary according to the amount of food assimilated. Death very occasionally occurs from pure inanition, sometimes from the supervention of tuberculosis. Sometimes the patient has regained the desire to eat and to live, but too late, the power to assimilate nourishment being lost. The author thinks it necessary, in view of the statements that have been made that hysterical anorexia is not attended with wasting, and is never fatal, to give some tables of cases, showing the daily loss of weight, and to emphasise the fact that the disease may undoubtedly, though rarely, have a fatal termination. Speaking generally, digestion is retarded, and the amount of HCl in the gastric juice deficient, but there are not at present sufficient data to state exactly the alterations in the gastric juice.

The most important affections of the intestines in hysteria are hysterical tympanites, meteorism, or phantom tumour, and spurious peritonitis. The author divides cases of meteorism into two varieties, according as the distension is general, affecting the whole abdomen, or localised. In both kinds, the difficulty of diagnosis may be very great. This subject is treated fully and well. As to the mechanism of production of hysterical tympanites, the localised form could be accounted for (Erbstein's theory) by local contractions of the intestinal fibres, whereby local production of gases is favoured, and is imprisoned when formed; or by the contraction of certain of the muscles of the abdominal wall. The generalised variety cannot be so explained. Brodie showed, by introducing a tube into the rectum, that when the gas was passed the abdomen became flattened; it soon, however,

swelled up again. But the tympanites has been known to disappear spontaneously, or under chloroform, without the passage of gas *per os* or *rectum*. Alternating paralyses and contractions of the intestinal walls may partly account for it; but the author thinks it difficult to admit Ebstein's view that there is insufficiency of the pylorus, and that carbonic acid gas and swallowed air passes from the stomach into the intestines. Others have supposed that the gases are derived from the blood, and are re-absorbed by the latter when a sudden disappearance of the distension occurs. In some cases hysterical tympanites simulates intestinal obstruction, even to the presence of fecal vomiting. In connection with this it may be mentioned that there is evidence that reversed peristaltic action may take place in hysteria, *e.g.*, an enema having been vomited soon after administration.

Hysterical disturbances of the urinary and genital organs receive full discussion. In considering cases of anuria, or ischuria, the author points out that attacks of ischuria of short duration are not uncommon in hysteria, and often pass unnoticed. In the more serious attacks the greater number have for their primary symptom constant vomiting, and the ischuria is merely secondary to this; sometimes, but rarely, ischuria is the primary, and vomiting the secondary affection. The vomitings must be held to be, to some extent, compensatory for the suppression or diminution of urine, as a constant relation is to be observed between the curves of the quantity of fluid vomited and that of urine passed, the one varying inversely as the other. In some cases where anuria or ischuria has occurred with vicarious phenomena, especially vomiting, the patients have gone as long as eight and seventeen days without uræmic accidents supervening. Rossoni has shown in two cases where there was ischuria, and not total anuria, that the administration of twelve grammes of urea in the one, and subcutaneous injection of fifteen grammes in the other, determined an attack of uræmia, though no marked symptoms of uræmia were previously present. The uræmic symptoms were relieved by pilocarpine, which, in the first patient, produced salivation, sweating, and urine containing urea; in the second, sweating and salivation only. The author sums up as follows:—In the large majority of cases hysterical anuria is accompanied by vicarious phenomena, such as vomiting, sweats, or diarrhœa. Cases of prolonged anuria or ischuria without such vicarious symptoms are very rare, and are sometimes accompanied by symptoms of uræmia, on whose gravity, in

the absence of a sufficient number of observations, it is very difficult to pronounce at present.

In the treatment of hysteria it is advised that if the mother suffer from confirmed hysteria, with symptoms of ordinary intensity, the children should be removed from home at an early age, and educated away from it. This is especially important in the case of girls, or, if the children have shown the premonitory symptoms of hysteria in the shape of night terrors, agitated sleep, or somnambulism. Such children are generally precocious, and require no stimulus to intellectual work; they should receive a sound physical training; the love of the marvellous and excess in amusement or in religious exercises should be especially discouraged. The influence of marriage is considered to be, speaking generally, favourable in female hysteria. In pronounced hysteria in men marriage is to be deferred, and the final advice to marry or not must depend on the disappearance or persistence of the hysterical symptoms. There is a useful section on the examination and anamnesis of hysterical patients.

With regard to hypnotism, the author urges the view held at the Salpêtrière. The hypnotic state is considered to be identical with an hysterical paroxysm, but provoked instead of arising spontaneously, and to act like hysterical paroxysms in profoundly modifying "*le terrain hystérique*." It may, hence, be dangerous even in experienced hands, and should be employed only when the accidents that may be excited by it are less grave than those which it is desired to cure.

It is hardly necessary to say that the importance of isolation in suitable cases is strongly insisted upon. Hydrotherapeutic measures are useful, especially the cold douche. Static electricity is recommended, especially for treatment of anæsthesia and muscular weakness, and as diminishing the frequency of the attacks; it also improves the general state of health and of the digestion, and acts as a tonic. Faradism is especially useful in the treatment of localised anæsthesias.

In the treatment of the convulsive attack, apart from taking precautions to prevent the patient injuring herself, no special measures are advisable. Should, however, laryngeal spasm or other urgent symptoms make it advisable to cut short the attack, the inhalation of chloroform in fractional doses, as in confinements, is the best method to employ. If the attack is the preliminary to a series of fits, or to a comatose state, chloroform is hardly suitable, and hypnotism may then be tried. In lethargic states the author advises to seek for a hysterogenic

zone, whose irritation will throw the patient into a paroxysm, such as is the common natural termination of such states.

In the case of contractures, they are, if possible, to be promptly dealt with ; they may be thus prevented in their incipient stage. If practicable, the antagonists of the contracted muscles may be thrown into contraction by stimulation with a faradic current, if anæsthesia be present, or with static electricity, if there is hyperæsthesia. If these measures fail, resort may be had to suggestion during hypnosis. Examination under chloroform of the contracted limb is of great value, in order that if adhesions have formed they may be broken down. When the patient comes round let the limb return to its former position if it tends to do so, M. Charcot's experience having shown that the trouble is only prolonged by fixing the limb in splints in a position opposite to that of the contracture.

In paralysis it is important to prevent contractions of the tendons, &c., arising from a faulty position of the limb ; when one limb is paralysed, movements of the sound limb may be usefully employed to call up the dormant sensations of the muscular sense in the paralysed one.

In persistent vomiting, with its consequent anorexia, and in primary anorexia nervosa, isolation is the essential point in treatment ; and the author quotes with approval M. Sollier's advice that immediately isolation is established the patient should be made, from the very first, to take ordinary meals. He has found no bad results occur from at once giving the patients solid food ; as a rule, they are able to digest it. It should be insisted upon that they eat, and recourse to forced feeding, either by the tube or by means of hypnotism, is undesirable.

The author has consistently endeavoured to make his book a practical clinical treatise, a complete embodiment of all hitherto observed hysterical phenomena. It is thus necessarily of great length, partly owing to the enormous mass of the literature on the subject. This literature, French, German, and English, is very fully treated ; French writings largely predominate, but some account will be found of nearly every observation of interest or importance from most sources. Quite a feature of the book, and one which adds greatly to its value as a work of reference, are the historical prefaces to each section, containing much interesting, and often very curious information. The various theories of origin of hysterical symptoms receive only brief notice, the aim of the work being the consideration of hysteria from the point of view of clinical observation, the description of its clinical manifesta-

tions. At the same time the neurosis is treated from the standpoint of the views held with regard to it by Charcot and his pupils, as the outcome of their long continued and laborious investigations on all its phases at the Salpêtrière. The book gives, therefore, a complete statement of what these views are, and, apart from any other considerations, would be interesting and valuable on this ground alone. Whatever the ultimate judgment on these researches may be they must always be remembered with gratitude as having done more than any others to place the study of hysteria on a rational basis, and to elucidate the many obscure problems underlying it.

According to this view hysteria is to be considered as characterised by (*A*) certain fixed signs—the stigmata, consisting chiefly of disturbances of sensation and of the special senses, together with special “diatheses” or tendencies; (1) to contractions “diathesis de contracture,” and (2) to muscular weakness, or amyosthenia, the latter in its most developed form finding expression in motor paralyses; and by (*B*) paroxysmal manifestations occurring as episodes on the groundwork of the stigmata of which the most common and most striking are the convulsive seizures. For these seizures a fully developed form is taken and considered to be the type, other paroxysmal disorders representing various phases of the typical attack. Hysteria is treated in this book from this point of view, all its manifestations being referred in their origin to the above characteristics. The plan of the work is thus first to consider generally the characters of the stigmata, in modifications of sensation and motion, then the paroxysmal symptoms, then the chief forms of contracture and paralysis, and, finally, the hysterical affections of the thoracic and abdominal organs, considered as expressions of one or more of the above general characters of the disease in one particular organ.

No doubt clearness is gained by treating a complicated subject on these lines, and the descriptions are simplified and systematised. The recognition of modifications of sensation, and of the special senses (these modifications having more or less peculiar characteristics), as very frequent symptoms, capable of easy identification in cases of hysteria, forms a step in advance in diagnosis. Perhaps the recent researches on the characters of hysterical anæsthesia are especially noteworthy as throwing light on the essential nature of the disease. At the same time the matter is complicated by the readiness with which the hysterical patient reacts to suggestion. Only in this way can the great variations

in the frequency of the occurrence of the stigmata, found by different observers, be accounted for. Such differences are to be partly explained by differences in the methods of examinations, and in the surroundings in which the patient is placed during observation.

A curious fact in the history of hysteria is that whenever a new combination of symptoms is identified as a clinical entity in neurology, cases of hysteria are sooner or later found, in which the newly-described group of symptoms is closely simulated.

It is, however, often difficult to trace out the analogy of symptoms in individual cases with the general type as represented in the above scheme. For instance, it is admitted that the complete attack of "hystero-epilepsy," as given in Charcot's masterly description, is of very rare occurrence, exceedingly so in comparison with the common milder attacks. And the convulsive attack commonly met with bears but a distant resemblance to the supposed complete form. It does not seem that anything is to be gained in simplification, but rather that still further complication is produced by describing the minor attacks in terms of the other. Similarly, in referring symptoms connected with the disturbance of a special organ to a hypothetical general type, assumptions are involved of whose accuracy we have no positive knowledge.

At the same time such a classification of symptoms, regarded as a provisional one, even if in the light of fuller knowledge it may in the future need considerable modification, is a decided aid to the study of the disease, in that it accentuates the fact that in dealing with hysteria we are dealing with a definite group of symptoms which result from a distinct and special disturbance of the central nervous system; and, although extremely diversified, present a certain constancy and order, and do not occur irrespective of all rule. It should be remembered that the investigations of Charcot were the first systematic attempt to bring order out of chaos. The author's observations on the modification of the urinary excreta during the various paroxysmal manifestations of hysteria, based as they are on a long series of carefully conducted investigations, deserve attention, and should be widely carried out. Such a result is of great interest, and if more extended experience confirms it, should prove a valuable aid in the diagnosis of doubtful cases. Mention must finally be made of the many excellent illustrations which adorn the book, and, again, to its value as a work of reference on the subject.

J. MICHELL CLARKE.

The Occipital Lobe and Mental Vision. — J. Soury, *Revue Philosophique*, January, February, December, 1895; February and March, 1896.

(A) FROM RETINA TO PRIMARY SUB-CORTICAL OPTIC CENTRES.

In these articles, M. Soury summarises the literature of "mental vision" from 1890—when he gave a critical account of its facts and doctrines in *Fonctions du Cerveau* (Paris, 2 ed., 1892). He discusses "(a) the cerebral centre of mental vision, (b) the intra-cerebral nervous apparatus relative to this function, consisting of primary optic centres (external geniculate bodies, pulvinar of optic thalamus, anterior quadrigeminal tubercles), and the projection and association bundles which unite the retinal fibres to the ganglia of origin for the optic nerve, and these ganglia to the cortex of the occipital lobe." The excitability of the cortex—recent histological methods (Weigert and Pal, Golgi, Ramón y Cajal, Ehrlich, Nissl)—the overthrow of the network theory of Gerlach and Golgi—the establishment of the theory of neurons, and propagation of nerve impulses by contiguity instead of continuity—as well as the comparative and virtually antagonistic development of basal ganglia and cortex of cerebral hemispheres along the scale of vertebrates—form the subject-matter of his critical digest. The following monographs are cited as the most important among the recent publications:—Munk's *Sehspähre und Augenbewegungen* (Berlin, 1890); Monakow's *Experimentelle u. pathologisch-anatomische Untersuchungen über die optischen Centren u. Bahnen nebst Klinischen Beiträgen zur corticalen Hemianopsie u. Alexie* (Arch. f. Psych., xxiv., 1892); Henschen's *Klinische u. anatomische Beiträge zur Pathologie des Gehirns* (Upsala, 1890-1892); *On the visual path and centre* (BRAIN, 1893); *Sur les centres optiques cérébraux* (Rev. générale d'ophtalmologie de Dor & E. Meyer, 1894); Wilbrand's papers (*infra*); Sachs on the occipital lobe, *Das Hemisphärenmark des menschlichen Grosshirns. I. Das Hinterhauptlappen* (Leipzig, 1892); Vialet's *Centres cerebraux de la vision et l'appareil nerveux visuel intra-cérébral* (Paris, 1893); and the anatomo-pathological studies of Dr. and Madame Déjerine. (Other references will be found in the bibliography at the end of this abstract).

Constitution of Optic Nerve.

The optic path as a whole runs from retina to cortex, but contains two parallel systems of conductors (a) centripetal, (b)

centrifugal in direction (R. y Cajal). The former arise in the axis-cylinder prolongations of the large cells of the retina, which form the optic nerve. They arborise freely with the protoplasmic expansions of the principal groups of nerve cells in the external geniculate bodies, and pulvinar of the optic thalamus. From the intercalary cells of these ganglia other axis-cylinder processes traverse the posterior end of the internal capsule, continue as the white sagittal substance, or optic radiations of Gratiolet, and end in the nervous felt-work of the fifth (and third) layer of the occipital cortex. There a new system of neurons (*neurones associatifs*) forms connection with the other cells of the cortex, more particularly with the giant solitary cells.

The centrifugal fibres start with the axis-cylinder processes of the pyramidal cells of the cortex, and arborise with the nerve cells of the superficial grey matter of the anterior quadrigeminal tubercles; the processes from these ganglia again terminate in the nervous felt-work of the retina (Monakow, 1892). The giant solitary cells of the occipital cortex are found to atrophy after section of the posterior segment of the internal capsule. Munk also affirms that centrifugal fibres are directed from the visual area to the centres of movement for the eye.

Reflex and Voluntary Movements of the Eye.

The experiments of Munk, Fritsch, Hitzig, Ferrier, Luciani, Tamburini, Bechterew, and Schäfer are summed up as follows:—(i.) The entire visual centre of one hemisphere is related with the homonymous halves of both retinae; (ii.) the superior zone of the visual centre of one hemisphere is related with the upper part of the homonymous halves of both retinae; the inferior zone with the lower part, the intermediate zone with the intermediate part. Schäfer also noted accompanying movements of the upper eyelid and pupillar modifications. Later on he showed that after removal of both frontal lobes anterior to the sylvian fissure, and section of the corpus callosum, electrical stimulation of the cortex still resulted in associated eye movements. The centre of these movements must, therefore, be sub-cortical, and probably lies in the grey matter of the quadrigeminal tubercles.

Danillo also noted conjugate deviation of the eyes, at the fifth month, in puppies and kittens, on exciting the occipital cortex (1888). Excitation of the white matter, after removal of the cortex, produces the same at the second month; equally so

if the cortex of the anterior motor region is removed, or isolated by vertical or horizontal sections. Excitation of the white matter of the motor zone produces no movement of the eyes. From this Danillo concluded that—(i.) The occipital lobes do not comprise centres analogous to those of the motor zone; (ii.) Ferrier's hypothesis (which accounts for the movements of the eye by subjective optic sensations, due to excitation of the occipital lobe) cannot be sustained; since stimulation of the white matter provokes the same movements, both after ablation of the grey matter, and at a period when the cortex is still inexcitable; (iii.) the phenomena observed after vertical and horizontal section of the brain, or extirpation of the motor zone, indicate that the centres of conjugate deviation lie deeper than either motor region of the cortex or occipital lobe. M. Soury suggests that the electrical stimulus in these cases may act upon preformed cerebral mechanisms, replacing the physiological nervous stimulus.

Munk established similar conclusions as to the independence of the eye movements caused by excitation of the occipital lobe; and since a horizontal incision, dividing the fibres of the corona radiata of the visual sphere, invariably blocked them, it is obvious that the oculo-motor functions of the occipital lobe are in relation with the basal ganglia *via* the optic radiations of Gratiolet. The occipital lobe can, therefore, no longer be regarded as purely sensory, seeing that its excitation provokes movements of the eye, upper eyelid, and iris. In contradistinction to the oculo-motor effects of visual sensation, due to excitation of the visual sphere, Munk determines other movements, which persist after ablation of both cortical centres of vision. He distinguishes (i.) *retinal reflexes*, or contraction of the pupil to light, even after removal of the cortex; (ii.) *visual reflexes*, e.g., winking, avoidance of obstacles; (iii.) *acquired visual reflexes*, e.g., flight from a whip, &c. Excitation of the occipital lobe induces reflexes of the second order. "We know," continues Munk, "that the corona radiata of the visual sphere contains, besides the fibres of the optic nerve, whose excitation, running centripetally to the visual area, determines vision, other fibres travelling in the same radiations, of which the excitation—starting with the visual sphere and passing centrifugally to the lower, sub-cortical regions of the brain—determines movement; and that the simple movements of the eye (as well as the movements of the eyelids, &c.), resulting from vision, are effected by this path; while other movements contingent upon sight need the inter-

polation of association-bundles and other cortical elements." These movements of the eye can, therefore, only be due to centrifugal impulses from occipital lobe to sub-cortical centres, *vid* corona radiata (Sherrington, 39). Monakow places these centres in the anterior quadrigeminal tubercles. Knies further refers the movements of convergence and accommodation, which he characterises as "voluntary," to the occipital lobe; so that its cortex is both sensory and motor. And, in fact, convergence and accommodation are affected in all cortical troubles of vision, while the inferior ("involuntary") ocular reflexes may persist.

Clinical evidence shows that lesion of the anterior quadrigeminal tubercles is not essentially injurious to vision, but that it does involve disturbance of eye-movements and of pupillar innervation (Monakow). In the lower animals, even in certain mammals, these ganglia are of main importance to vision, as compared with the external geniculate bodies and pulvinar of the thalamus. The fibres which they contribute in man to the optic tract are relatively insignificant (Monakow, Bernheimer). The *pupillar luminous reflex*, however, appears to be governed by these bodies.

Argyll Robertson was the first to show (1869) that this reflex (direct contractility to light) can be abolished; while movements of accommodation and convergence are preserved. The fibres of the optic nerve transmit the luminous stimulus which governs movements of the pupils, to the point at which originate the branches from the oculo-motor nerve that innervate the sphincter of the iris. Wernicke finds that the reflex arc between retina and oculo-motor nerve, does not extend beyond the quadrigeminal tubercles, since (i.) when the path of the pupillar fibres is interrupted behind these ganglia, the reflex arc is left intact, and the pupils give nominal reaction to light; (ii.) when it is interrupted in front of them, the pupillar reflex fails to appear. Mendel's detailed experiments point to the *ganglion habenulæ* as the precise centre of the reflex. Iridectomy seems invariably to produce atrophy of this organ. He, accordingly, defines the path of the reflex as optic nerve and ganglia of habenula on same side, thence by posterior commissure to nucleus of Gudden, and so to fibres of oculo-motor trunk. It is to be noted that the loss of the pupillar reflex occurs in fifty to sixty per cent. cases of general paralysis, in which lesion of the ventricles, more especially of the third, is a common feature. The expression of the emotions shows the same anatomical and functional independence, accord-

ing as the facial movements are reflex or voluntary in origin. In the latter, the nervous impulses travel *via* internal capsule, and the foot of the cerebral peduncle; in the former, *via* optic thalamus and tegmentum of the peduncle. The loss of the emotional involuntary reflex of the facial nerve depends upon lesion of a sub-cortical ganglion (optic thalamus, or fibres of corona radiata); voluntary movements, such as mimicry of the emotions, are inhibited by lesions of the cortical centre of the same nerve.

Functions of Cerebral Cortex in the Scale of Vertebrates.

Passing on to the deductions made by Edinger (10), and by Goltz himself (15), from the effects of Goltz's operations on the cerebrum, M. Soury denies that any luminous sensation, or perception, can exist after the cortex has been totally extirpated. He refers the facts adduced to survival of sub-cortical centres, and to reflex actions usually associated with visual sensation, but intrinsically independent of it. Such are the pupillar reflex, and the *Blendungsreflex*, cited by Goltz.

Edinger's preparations plainly show the lacuna which exists between the dog's brain and that of man, or even the monkey. In the latter, minimal destruction of the Rolandic cortex determines paresis or paralysis—impotence of voluntary movement, and troubles of sensibility; in the dog (as in lower vertebrates) loss of the cortex does not imply loss of co-ordinated movements, nor of such as are voluntary or acquired, *i.e.*, represented in the cerebral cortex. Soury further pleads that Goltz's dogs are in a state analogous to the dementia of paralysis, and are, therefore, of no account in the *pros* and *cons* of cerebral localisation. In man, he concludes, (as opposed to lower vertebrates), total destructive lesion of the cortex of both occipital lobes produces absolute cecity: man cannot see with his lower, sub-cortical optic centres.

Trophic Centres of Optic Nerves.

The direct trophic influence of the retina upon the optic nerves is well-established. In cases of anophthalmia, the optic nerves, chiasma, and fillet, are wanting, the occipital convolutions on either side being atrophied. In subterranean animals, whose visual apparatus has degenerated, the tactile sense is peculiarly responsive to luminous excitation. Dubois (7) reduces the mechanism of vision, in the last resort, to a tactile manifesta-

tion. The syphon of pholas dactylus contracts to light, its photodermic functions resembling those of the retina.

Connection of Retina with Primary Optic Centres.

The histology of the retina and visual fibres are considered under this heading, but must here be omitted. The visual acuity of the *macula lutea* is said to depend upon the delicacy with which each cone (7,000 to 7,500) transmits its excitation to a bi-polar cell, this again to one protoplasmic arborisation of the ganglionic layer. The autonomy of the macular region persists throughout the optic nerve to the cortical centres; and the preservation of central vision in the majority of cases, where (as in hemianopsia) the entire visual cortex is destroyed on one side, indicates that each macula is connected with both hemispheres. The line which separates blind half from normal half of the field is not median; it passes five to ten degrees beyond the centre, leaving the macular region intact.

The importance of the sub-cortical centres must be remembered. They are the ganglia of origin for the optic nerve—the post-house where impressions conveyed by the retinal fibres are transmitted to other couriers. The transmission of visual impressions must be considerably modified by the distribution of the retinal fibres in the basal ganglia. An axis-cylinder process may interlace with one or with several dendrites of the nervous layer, which again reacts upon the cortical distribution.

In re centrifugal fibres, M. Soury adduces, as physiological evidence of their existence in the optic nerve, the discovery of Engelmann (11) of the movements of the cones and pigment of the retina under luminous and nervous influences. The cones of the eye retract in light. In a “dark” frog, direct illumination of one eye causes the other to contract. When the brain has been destroyed, light only affects the eye directly illuminated. This functional sympathy between the two retinæ can only be explained by synergic association of the cones through the optic nerves, acting centrifugally instead of centripetally. Hence Engelmann’s hypothesis of two kinds of fibres in the optic nerve—(a) sensory, centripetal, conducting luminous and chromatic sensations; (b) centrifugal, motor, or “retino-motor.” Indirect excitation of the optic nerve by strychnin produces the same action on the retina. Additional evidence is contributed by Nahm-macher’s experiments on amphibia (1893), Gotch and Horsley’s on the spinal cord (1892.)

(B) FROM PRIMARY OPTIC CENTRES TO CORTEX OF OCCIPITAL LOBE. CALCARINE REGION AND CORTICAL RETINA.

M. Soury adopts the position of Henschen in regard to the cortical centre of vision. "Le lobe occipital est le centre cérébral de la vision mentale. De la rétine aux centres primaires optiques—corps genouillé externe, pulvinar de la couche optique, tubercules quadrijumeaux antérieurs—et des centres primaires optiques à l'écorce du lobe occipital, des faisceaux de projection relient les fibres rétiniennes,—nerfs optiques, chiasma, tractus,—aux ganglions d'origine des irradiations optiques, dont les fibres se terminent par des arborisations libres sur la face interne du lobe occipital, dans le territoire de la scissure calcarine, véritable rétine corticale." The visual area is much more extended than the centres for perception of sensations of light and colour; and very different theories obtain as to the functions of those regions of the occipital and parietal lobe which it comprises. The anatomoclinical method (Monakow, Henschen, Déjerine, Sachs, Vialet, Redlich) throws more light on the functions of the nerve centres than the earlier anatomical or purely experimental researches. Thus it has been shown that in cases of blindness, involving widespread degenerations, the geniculate bodies alone are in direct relation with vision. The fibres of the optic nerve which pass into the pulvinar of the thalamus, the anterior quadrigeminal tubercles, the temporal and parietal lobes, are not visual (Henschen).

Degeneration of the pulvinar does not produce hemianopsia, when the geniculate body is intact. The anterior quadrigeminal tubercles may submit to grave lesion without disturbance of vision. The optic fibres in these ganglia must be accessory to the mechanism of vision; they do not conduct luminous and chromatic impressions from retina to cortex. The pulvinar and quadrigeminal tubercles are possibly reflex optic centres—in man they probably do not contain a single visual fibre. The geniculate bodies alone are in relation with vision, and lesion of these ganglia inevitably causes hemianopsia. Certain forms of sub-cortical hemianopsia may indeed be accompanied by pulvinar lesion, but the actual cause of cecity is lesion of the external geniculate body. Destruction of this ganglion determines an almost total disappearance of the bundle of Gratiolet, through which pass the visual fibres from the ganglion. So, too, in retrograde degeneration, the partial destruction of the occipital lobe may determine atrophy of the internal capsule, yet the fibres

involved are not visual. Lesions of the occipital lobe react upon the retro-lenticular region of the internal capsule—the fibres of which end in the central grey nuclei, while the capsular fibres proper pass on into the cerebral peduncle. (Déjerine.)

Clinical application of these conclusions is not slow to follow. Pick recognises that a lesion exactly limited to the optic thalamus produces no alteration in perception of colour and form, nor any deficit in the visual field. Lesion of the external geniculate body, or optic radiations, is required to provoke these troubles. The symptoms which originate from the thalamus are quite different, *e.g.*, motor troubles of expression—imitative, involuntary, automatic, &c. In man, the anterior quadrigeminal tubercles are equally deprived of the importance which Griesinger attributed to them in vision. Recent pathological observations prove that lesions of these ganglia may develop without essential alteration of vision; they involve motor troubles of the eye, and of pupillar innervation. The optic lobe in man is no more than a reflex centre. As we have seen, it is still (without speaking of the luminous pupillar reflex) of prime importance in the transmission of centrifugal impulses (*supra*, Wernicke, Monakow, R. y Cajal).

Nor has the inferior parietal lobule, or angular gyrus, any more direct relation with vision. Ferrier's localisation of the cortical centre of distinct vision in the angular gyrus of the opposite hemisphere—Charcot's explanation of the crossed unilateral amblyopia of hysterical hemianæsthesia, by a complementary decussation of the direct bundle of optic fibres, are, therefore, of no more value than the early observations of Goltz, Ferrier, and Munk, to the effect that unilateral lesion of the visual centre in dog and monkey determines complete cecity of the opposite eye, and not partial cecity of both retinae—a conclusion based on the theory of complete decussation in these animals.

It was reserved for Henschen¹ to point out the true analytic method. "When the visual area is removed, the fibres which conduct the luminous impressions degenerate; secondary changes also occur in the fibres which unite the visual area with other portions of the cortex and with the central ganglia." In a case of hemianopsia, *e.g.*, it is illegitimate to charge with the visual disturbance all tracts that are found degenerated. Negative must be opposed to positive cases, and the principle of elimination, as laid down by Exner, Séguin, &c.,

¹ Henschen. "Sur les centres optiques cérébraux," p. 3, Extrait de la *Rev. génér. d'Ophthalmologie*, No. 8, 1894.

must ultimately result in the discovery of one constant, invariable lesion in cecity. Henschen,¹ by careful clinical comparison, arrived at the *cas type*, and determined the inevitable lesion to be localised in the cortex of the calcarine fissure. The other lesions observed have only produced blindness, inasmuch as they involved the visual fibres of the radiation of Gratiolet on its way to the calcarine cortex. The optic centre cannot, therefore, be localised on the external surface, or convexity, of the lobe—as determined by Munk and other physiologists: such lesions as they diagnosed must have involved the optic radiations. In Henschen's words, "hemianopsia results from a lesion only when it destroys the calcarine cortex, or the optic bundle which unites the geniculate body to this part of the occipital lobe. This bundle occupies the inferior part of the radiation of Gratiolet."

In Henschen's typical case, in which the patient was an intelligent subject, not suffering from dementia, the lesion was a softening produced by thrombosis, strictly confined to the cortex, lying deep in the calcarine fissure, and not involving more than one or two mm. in depth of the subjacent white substance. The case was not complicated by any lesion of the central ganglia. Microscopic investigation only revealed a secondary degeneration in the optic radiations, in consequence of the cortical softening.

The anatomical structure of the calcarine cortex (though differing from the rest of the occipital lobe, not merely in the thickness of the molecular layer, but also in its development of horizontal fibres, forming the band of Vicq d'Azyr), is not special to the fundus of the fissure. The same band extends for several mm. along the two lips of the calcarine fissure, the superior of which belongs to the cuneate, the inferior to the lingual lobe. It is to be expected that such identity of structure in either convolution should result in identity of function. And Hun adduces a case in which atrophy of the superior lip of the calcarine fissure produced hemianopsia of the inferior quadrant of the visual field on both sides. Wilbrand shows that the lower lip corresponds with the upper visual field. There would thus appear to be projection of the retina upon the cortex of the occipital lobe, as contended by Munk, and disputed by Monakow. Henschen accepts it, and proposes the name of "calcarine retina" for that part of the cortex of this fissure at which, according to him, there is projection of the elements of the peripheral retina, *via* the visual fibres of the optic nerves, chiasma, tracts, and optic radia-

¹ Cas de Henschen-Nordensen. "Klin. u. anat. Beiträge zur Pathologie des Gehirns," II., p. 387. Obs. XL.

tions. The macular bundle, though lateral in the papilla of the optic nerve, is certainly central in the chiasma and tract. Beyond the external geniculate body, however, Monakow's anatomical objections appear to be well-grounded. Vialet proposes a physiological shortening of the path of the visual impulses, due to association, so that a ganglion cell of the retina might always be in relation with the same cell, or group of cells, of the cortex, owing to the incessant repetition of the transmitted impressions. The central and peripheral parts of the cortical retina would lie, according to Henschen, in the anterior and posterior parts of the cortex of the calcarine fissure. Sachs, again, has a case which does not correspond with this somewhat restricted cerebral localisation of the *macula lutea*. Henschen, like Wilbrand, admits that each half of either *macula lutea* is in connection with both cerebral hemispheres, while the macular fibres also undergo partial decussation in the chiasma, and are divided into direct and crossed bundles.

The preceding observations refer to the "cerebral retina," and not to the much larger region of the "visual area," which is the anatomical substrate of mental vision. While the region on which visual impressions are projected is very limited, that corresponding with visual representations is vast, and may very likely involve the convexity of the occipital lobe and the angular gyrus. Henschen concludes that the seat of perception and of representation occupy distinct positions on the occipital and parietal lobes. Vialet distinguishes "a visual centre of perception" and "a visual centre of memory," explaining their relation by association bundles. Déjerine localises the "visual centre of words" in the angular gyrus.

Nothing is certain, however, beyond the fact that there is a cortical centre of visual perception, and that its lesion entails cortical hemianopsia. As to psychical cecity there are no proofs; it is a question of interpretation. At this point, Munk's dual schema of the cortical elements of perception and representation gains general acceptance, but simply as a *pis-aller*, because no other explanation is to hand, *e.g.*, in cases where the patient suffers from verbal cecity, loss of intelligence of written signs, along with unimpaired vision of the same signs. It is thus easy to pile up distinct centres of memory for all external objects. Vialet regards it as certain that some cerebral elements gather up new perceptions, while others warehouse the visual memories. M. Soury urges, on the contrary, a more sceptical attitude until further clinical evidence shall be forthcoming.

He sums up by defining the "optic path," or system of visual fibres properly so called, as constituted by two neurons—(1) an *anterior neuron*, formed by the great ganglion cells of the retinae and their prolongations, the elements of the bundles of the optic nerve, the chiasma and optic tract, the terminal axis-cylinder ramifications, arborising in the protoplasmic branches of the cells of the external geniculate body; (2) a *posterior neuron*, formed of the cells of the external geniculate body and their prolongations, which, as fibres of the inferior portion of the optic radiations, arborise in the middle of the different layers of nerve cells in the cortex of fundus and lips of the calcarine fissure.

The Occipital Lobe.

Space forbids us to enter fully upon M. Soury's review of the comparative morphology, arterial supply, embryology, and histology of the occipital lobe. This lobe is small in man as compared with the rest of the hemisphere; in monkeys it is highly developed. On the other hand, it is in man (unlike the monkey) as highly convoluted as the rest of the cerebrum; hence the superior size of its surface, no doubt in relation with the increased number of fibres from the optic radiations which spread over the calcarine region, and effect a high elaboration of central visual impressions. The external convex surface presents no connecting gyri between the occipital and the parietal and temporal lobes. So, too, the internal surface is continuous with the temporal lobe. The external parieto-occipital fissure is very small. In microcephalous cases (= pure arrest of development — idiocy) it is extensive; while the external connecting gyri become once more pronounced, and the occipital lobe is smooth, recalling that of inferior monkeys.

M. Soury does not admit the transverse occipital sulcus to be the homologue of the "*sulcus du singe*," as held by Ecker and Rudinger. In man this sulcus divides the occipital lobe into the superior and inferior regions. Where the "*sulcus du singe*" really exists, it seems, again, to imply arrest of development.

On the internal surface the calcarine fissure appears in the first months of intra-uterine life as two branches, which subsequently unite with the parieto-occipital fissure to enclose the cuneus. (In micro-cephalics the cuneate lobe is small and smooth, while the *pes hippocampi minor* is often wanting. In

Chinese brains, the cuneus appears to be very small). The calcarine fissure above, and the collateral fissure below, enclose the lingual lobe. Between the collateral sulcus and third temporal fissure lies the fusiform lobe. The cuneus, lingual lobe, and fusiform lobule are the three convolutions of which the lesions appear to account for hemianoptic symptoms, and which many clinical authorities regard as the cortical centre of vision.

These regions of the brain are supplied by the posterior cerebral artery; in particular by its two posterior branches, the posterior temporal and occipital arteries. The latter ramifies over the calcarine fissure, and, under the name of calcarine artery, is responsible for the nutrition of the most important regions of central vision. Monakow remarks that the obliteration of this artery is especially provocative of lesion, because it induces softening of the cuneus. The occipital artery may thus be termed artery of cerebral vision, or hemianoptic artery. The obliteration of any one of its branches, in particular of the calcarine artery, induces a lesion (anatomical and functional) of the cuneus and surrounding regions, especially of the lingual lobe. In arterial thrombosis, the seat of the softening is often less extensive in the cortex than the vascular region; because there is a possibility of collateral irrigation from neighbouring vessels, since the cortical arteries are not terminal, and anastomose freely (Mendel). In the white substance and basal ganglia, on the contrary, the arteries are terminal. Monakow insists on the gravity and complexity of troubles in the visual apparatus, arising from even a restricted area of softening of the white matter of the cuneus, since this involves the destruction of projection fibres passing from the primary optic centres of the convolutions of the convexity and lingual gyrus. It is more especially in regard to vascularisation in the central nervous system that the connections of its respective parts are of such importance; for the destruction of a nerve centre by anæmic softening or hæmorrhage, or the mere slowing-down and progressive extinction of its activity by arterial obliteration (local anæmia), reacts infallibly on the regions of the brain connected with this centre. The innumerable neurons which come from all points to terminate here, whether by their collaterals or by the arborisations of their axis-cylinders, as well as the dendrites or cellular bodies of the neurons of the affected centre, undergo, in consequence of the disturbance of their normal activity, alterations in nutrition, so profound as often

to reduce to silence considerable, and sometimes distant, regions of the cerebral cortex. M. Soury queries the use of the term functional lesion (in cases where the original lesion cannot be detected on examination of the cortex); all functional alteration may, in the last resort, be reduced to a disturbance of nutrition, taking nutrition to mean the most general cause of modification in the structure and texture of the anatomical elements. He contends that the key to an interpretation of the functions of the occipital lobe—*i.e.*, to an understanding of mental vision—lies in the anatomy of its white matter, and connections with the neighbouring regions of the parietal and temporal lobes. The anatomical and functional relations of these lobes are of the utmost importance to the study of optic aphasia, verbal cecity, and alexia. In the anatomical descriptions that follow, Sachs (36) and Vialet (43) are the principal authorities quoted.

In describing the *forceps corporis callosi*, Sachs postulates the existence of at least two nerve paths. If the cortical region of distinct vision in both eyes, corresponding with the macular region of the retina, is represented in the two occipital lobes, the two cortical points of distinct vision must be commissured by the fibres passing through the forceps. Again, the right occipital lobe must be associated directly with the left temporal lobe, in order that objects seen in the right half of the visual field may evoke the corresponding auditory verbal image localised in the left temporal lobe. This path (according to Freund) must be interrupted in optic aphasia.

In the histology of the occipital lobe, Soury adopts the 6 cortical layers of Ramón y Cajal (as against Meynert's 8). These are—(1) molecular layer; (2) layer of fusiform vertical cells; (3) layer of median myeline cells, or striated band of Vicq d'Azyr; (4) middle pyramidal layer; (5) giant pyramidal layer; (6) layer of polymorphous corpuscles.

A schema of the visual path to the occipital lobe (after Monakow) again presents the idea of retina, basal ganglia, and occipital cortex as optic centres for the departure and terminus of systems of optic fibres—"systems that are highly solidarified, since disturbances of nutrition, defective lesions, react from cortex upon primary optic centres, and from these centres upon the cortex." The schema accordingly includes two sensory and two motor neurons, with the cortical association cells of the external molecular layer.

In this layer (as elsewhere in the cortex) are the giant pyramidal cells, which receive the impact of nervous commotion,

and ultimately (by the process of muscular adjustment) adapt the organism to its environment—internal to external conditions. Nervous impulses pass to these cells from (1) cells of Golgi, with short axis-cylinders; (2) association cells between the several lobules of one hemisphere; (3) cells of the opposite hemisphere *viâ* callosal fibres; (4) sensory (visual) cells; (5) cells of different layers of the cortex, arborising in the molecular layer.

The same molecular layer gives origin to “the motor reactions, still occasionally termed *voluntary*.” M. Soury points out that this word no more explains these reactions than does *consciousness* explain the vital processes of nerve-cells that have reached a certain intensity and duration. The nervous discharge, of internal or external origin (produced by the relative intensity of one group, or several groups, of associated mental images, or from the application to some region of the cerebral cortex of mechanical, chemical, or electrical stimuli), provokes, or arrests, muscular contractions of corresponding magnitude or intensity—actions or inhibitions, according to the nature of the centres excited. In sleep, or in chloroform narcosis, the motor reaction follows fatally the nerve-paths of least resistance, most worn, most frequently traversed; and the effect thus determined is inevitable. In narcosis the reaction is certainly unconscious; it is more or less conscious in dreams; in the waking state it is altogether conscious, though it varies with the nature of the act. But this epiphenomenon, consciousness, can no more modify the arrest, or production, of phenomena of central innervation, than the shadow of the traveller alters the rhythm or direction of his steps. The impulses transmitted from the sensory neurons to the dendrites of the great pyramidal cells of the molecular layer of the cortex, are the effective incitation of voluntary movement (R. y Cajal). In physiological excitation of the cortex, the stimulus may act directly on the dendrites of the pyramidal cells, or indirectly *viâ* nervous fibrils from the superficial layer of the cortex. In either case the artificial stimulus of physiological experiment acts on the highest nerve centres of the brain exactly like “the will.”

HEMIANOPSIA.

The characteristic symptom of lesions of the cortical centre of vision is homonymous, bilateral hemianopsia. Lesions of the right occipital lobe abolish function in the right half of each retina; the left half of the field of vision is blotted out; and *vice versa*. The sense of light or of colour may be affected. Hemidyschromatopsia (loss of perception of some colours), or hemi-

achromatopsia (loss of all colours) may precede hemianopsia in the halves of the visual field affected. In ophthalmic migraine, where the transitory disturbances (hemianopsia, cortical cecity, aphasia, anæsthesia, paresis, &c.) appear to be determined by equally transitory states of anæmia in the occipital and temporal lobes, the loss of colour-sense may precede the loss of perception of light and space. The transitory scotoma of ophthalmic hemicrania is a central disturbance (hallucinatory) from the cortical centre of vision, although the patient "sees black," a peculiarity formerly supposed to distinguish sub-cortical from cortical hemianopsia (Mauthner, Dufour).

Lesion of the internal surface of the occipital lobe inevitably produces cecity in the corresponding half of both retinæ, *i.e.*, in both eyes, never a crossed, monocular cecity of the opposite eye (Wilbrand, 1881). Ferrier is, therefore, disallowed (when he accounts for crossed amblyopia by saying that if the occipital lobe is in relation with the homonymous halves of both retinæ, the angular gyrus is more particularly in relation with the yellow spot of the opposite eye), and is rendered responsible for all later errors with reference to the functions of the angular gyrus, crossed amblyopia, &c., of both French and English writers. Nor was Charcot more successful in explaining the crossed cecity of hysterical hemianæsthesia. He assumed a second decussation of the direct bundles of the optic fillet (that did not cross in the chiasma), so that the optic nerve would cross over completely before entering the brain, whereby the unilateral lesion of one hemisphere would produce cecity of the opposite eye. His other theory, that in common sensory anæsthesia, the special sensory anæsthesia which is its usual concomitant in hysteria and other neuroses, as well as in certain organic affections of the brain, derives from lesion of the posterior segment of the internal capsule, is also invalidated on anatomical grounds (Bechterew).

Charcot's clinical facts (as gathered up by Féré, 12) remain for explanation. They may be summarised as follows:—

(1) The intensity of the amblyopia is always proportional to the intensity of anæsthesia in the eye; it is less pronounced when the conjunctiva alone is insensible; more pronounced when the cornea is equally anæsthetic.

(2) If the hemianæsthesia merely affects the extremities and leaves the face intact, the visual disturbance may be absent; but if the face is affected and the extremities intact, there will be amblyopia.

(3) If in hysterical hemianæsthesia, the hemianæsthesia can be suppressed, the amblyopia disappears also.

The same facts apply to the crossed amblyopia consequent on organic lesions of the brain.

Bechterew notes the relation between general anæsthesia and weakness of sight on the same side, or common and special sensory anæsthesia, and extends it even to hypnosis. Frankl-Hochwart noted that in hysteria, unaccompanied by troubles of sensibility, the visual field was usually normal, together with visual acuteness and perception of colour (which, however, might be slightly diminished). It is especially in anæsthetic hysteria that the visual field is concentrically reduced, and the perception of light and colours lowered. There is an evident relation between functional troubles of the eye and disturbances of its sensibility. Knies, in his "*Troubles centraux unilatéraux de la vision dans l'hystérie*," described the troubles that accompany general hemianæsthesia, including the eye and its annexes (cornea, conjunctiva, &c.), as a diminution of central visual acuteness, nearly always implying concentric reduction of the visual field, and alteration of chromatic sensibility; the functions of the eye being normal, and the state of the pupils variable, with no known corresponding anatomical lesion. These troubles are subject to modification by psychical influences. The chief objection to a cerebral origin of such unilateral troubles is anatomical. Above the chiasma, no lesion of conductors or optic centres is known that can determine unilateral visual troubles. Hence the long acceptance of Charcot's posterior chiasma. Knies offers a new hypothesis: the proximate cause of these unilateral troubles of vision in hysteria is to be localised in the peripheral optic apparatus; the effective cause is central—*viz.*, a cerebral disturbance of vascular innervation.

All brain activity is accompanied by vaso-motor processes, which are vaso-constrictive in nature. The ablation of a cerebral hemisphere is followed *inter alia* by hemilateral paralysis of the cervical sympathetic. In hysteria the loss of the vaso-constrictive action, exercised normally by the centrifugal nerve impulses of the cortex, determines vaso-dilatation of certain peripheral regions. The calibre of the vessels enlarges, and since this occurs at the *foramen opticum*, the nerves undergo mechanical compression. This is translated into disturbance or transitory abolition of condition, and of the function of the sensory organ connected. Such compression of the myeline of a nerve may produce abolition of its conductivity without destroying the axis-cylinder. When the vascular dilatation ceases, the effect, *i.e.*, the functional trouble, disappears also. Hysteria would, therefore, according to Knies,

be "a disturbance of vaso-motor innervation, of cerebral origin." Many other hysterical symptoms can be explained from the same cause, as well as hypnosis, suggestion, sleep.

Bechterew concluded that amblyopia depends upon anæsthesia of the ocular capsule. Section of the sensory or ascending root of the trigeminal (which induces anæsthesia to touch and pain in half the face and head, with amblyopia of the anæsthetic side, and diminution of sensibility in other sense organs on the same side), proved that amblyopia can be induced by lesion of a sensory nerve, of which the roots are distributed to the face, and more especially to the ocular capsule. In other organs, too, diminution of function is related with anæsthesia of the surface. This is the explanation of amblyopia, and sensory anæsthesia in general, in cases of unilateral anæsthesia of the face and sense-organs of cerebral origin—at least, in cases of lesion of the cortex and posterior third of the internal capsule, provided the special conductors to the sense-organs (optic nerve, acoustic nerve, &c.), and their cortical and sub-cortical centres, are not affected.

The detrimental influence of facial anæsthesia on the sense-organs arises from troubles of nutrition. The nutrition of any organ is imperfect unless the sensory and motor nerves distributed to it, and especially the vaso-motor nerves, are in a normal state.

Any interference, central or peripheral, produces the same effect. Duval and Laborde proved that lesion of the ascending root of the trigeminal affects nutrition in the eye; Lannegrace shows that cerebral lesion produces a similar disturbance. All interference with nutrition in a peripheral sense-organ is translated into a corresponding functional alteration, *i.e.*, alteration of sensation; and Bechterew refers such disturbances of nutrition to inadequate arterial irrigation. Hence the capital importance of the state of the vaso-motor system to the interpretation of amblyopia, and of sensory anæsthesia in general, in sensory hemianæsthesia.

The acuteness of perception, far from diminishing, may be augmented, if the afflux of blood is considerable in the organs of the special senses, or in surfaces of the skin and mucosæ serving for the perception of tactile, painful, or other excitations. We know that contraction of the vessels from cold diminishes the perception of cutaneous excitation to the point of anæsthesia, while it rises to hyperæsthesia under the action of causes which dilate the vessels (heat, sinapism, &c.). Sensations of cold, absence of sweat, analgesia, defective hæmorrhage after profound pricks in

hysteria, are, like anæsthesia, explained by the diminution of the finest arterial vessels of the surface of the skin. The reality of this vascular spasm in hysterical hemianæsthesia is attested by the loss of heat on the insensitive side of the body, by the greater resistance offered to the electrical current, &c. In artificial anæsthesia, centrally provoked, the cutaneous regions rendered insensitive are lower in temperature than the surrounding regions.

Anæsthesia of the skin and mucosæ produces retinal anæsthesia, amblyopia of the eye on the corresponding side, by depressing the nutrition of the anatomical elements which receive external stimuli of luminous or chromatic sensations, and this solely in virtue of vaso-motor troubles, caused by inadequate irrigation of the peripheral organ of vision. Bechterew's theory¹ (which is extended to other sense organs) explains the crossed amblyopia of symptomatic hemianæsthesia in certain neuroses, or organic affections of the brain, without implicating the optic conductors, primary optic centres, or calcarine region of the occipital lobe. Disturbance of vaso-motor innervation, anæmia of the peripheral organ of vision (in consequence of cutaneous anæsthesia extending to this organ, as to other sense organs), is the cause of sensory anæsthesia, and it is superfluous to invoke either Charcot's "*carrefour sensitif*," or the equally erroneous hypothesis of a complementary decussation of the direct bundles of the optic fillets, behind or within the quadrigeminal tubercles.

Central hemianopsia is never monocular. Monocular hemianopsia, temporal or nasal, may result from lesion due to compression of the internal or external sides of the visual fibres of the optic nerve, previous to their partial decussation in the chiasma. This lesion involves the pupillar fibres, and the pupil of the corresponding eye reacts feebly or not at all to direct illumination, while it reacts synergically to illumination of the other non-affected eye, since the reflex paths are intact on the side of the uninjured nerve, as well as the centrifugal path from the reflex centres to the iris of the affected eye—this is the consensual reaction of both pupils. The pupillar fibres, which originate in the retinæ, are distinct from the visual fibres in the optic nerve and chiasma, while they are not to be found in the occipital optic radiations. It is a moot point whether they pass into the optic tract. The pupillar reflex to light persists in

¹ Bechterew. "Ueb. die Wechsel-beziehung zwischen der gewöhnlichen u. sensoriellen Anästhesie (Functionsabnahme der Sinnesorgane) auf Grund klinischer u. experimenteller Daten." *Neurol. Centralbl.*, 1894.

total, or cortical, cecity, *i.e.*, double or bilateral hemianopsia, due to lesions of both occipital lobes. Henschen finds pupillar fibres in the optic tract up to the level of the external geniculate body, which they do not enter; the hemiopic pupillar reaction is not therefore provoked by lesion of the external geniculate body. Bechterew, on the other hand, holds that the pupillar fibres diverge from the tractus as early as the chiasma, to enter the wall of the third ventricle.¹ The crossed cecity determined in birds by the destruction of one of the optic lobes, leaves intact the contractility to light of the pupil of the blind eye. Before entering the optic lobes with the visual fibres, the pupillar fibres, therefore, divide off from the tractus, and go directly to the region of the nuclei of the 3rd nerve. Bechterew found that both pupils reacted to light after section of the optic tract, in mammals. In man, he affirms the persistence of the pupillar luminous reflex in cases of cecity due to destruction of the quadrigeminal tubercles by compression from a tumour of the pineal gland. In general paralysis and tabes, the pupil may have lost the luminous reflex, while preserving the reflexes of accommodation and convergence, neither vision nor movements of the eye having undergone any alteration. The pupillar fibres have thus from a certain point a separate path from the visual fibres, from which they are throughout absolutely distinct. Darkschewitsch and Mendel, Edinger, Bechterew, Flechsig, Bogrow, have as many theories as to this path. Nothing seems certain except that two bundles of fibres pass from the optic tracts to the central grey matter of the third ventricle, and may be in relation with the nuclei of the ocular-motor nerve.

Wernicke's hemiopic pupillar reaction comes under the category of hemianopsia. Pick gives the reflex arc of the luminous pupillar reflex as composed of a *reflex centre* (no doubt localised in nuclei of ocular-motor nerve), and a *centripetal path* (optic nerve, chiasma, tract, quadrigeminal tubercles, and nuclei of the 3rd pair). If the pupillar fibres, which effect the luminous reflex of the pupil, are simultaneously attacked on the homonymous sides of both retinae, by lesion of the visual fibres of the optic tract, and the two anæsthetic halves of the retinae are illuminated (avoiding diffusion of light), the pupils will not contract; if the light falls on the sensitive halves of the retinae, the pupillar reflex comes off. This is the hemiopic pupillar reaction.

¹ *Ib.* "Ueb. Pupillenverengernde Fasern." *Neurol. Centralbl.*, 1894, p. 802.

By compressing the chiasma at different points it is possible to produce *bi-temporal hemianopsia*—loss of function in the crossed optic bundle; *nasal hemianopsia*—lesion of the external or direct bundle; *superior and inferior hemianopsia*—compression above or below. Atrophy of the visual fibres by compression of the chiasma may also result from internal hydrocephalus.

Pierre Gratiolet, in 1854, discovered the central origin of the optic nerve. Meynert accepted his conclusions. Wernicke worked them out *in extenso*. Panizza, 1855, also pointed out that the bundles of fibres issuing from the posterior cerebral convolutions contributed to form the optic nerve, but he was less acute in his observations than Gratiolet; and Gudden, Luciani, Tamburini, all showed that the decussation of the optic nerves is only partial. Goltz admitted, in 1876, that each cerebral hemisphere was related with both eyes in the dog. He argued, however, that the visual disturbance consequent on operation was hemiamblyopia, or cerebral weakening of vision, and not hemianopsia. Munk was slow to admit bilateral disturbances of vision from lesions of the occipital lobe. He long held that the dog differed in this respect from the monkey, though eventually he established the relations of either hemisphere with both retinae in birds as well as mammals. Vitzow, Gudden, Ganser, followed in the same lines. Monakow (31) really determined the nature and conditions of bilateral homonymous hemianopsia. He showed by experiment that ablation of a cortical centre (Munk's visual sphere) reacted on the development of fibres and primary nervous centres connected with it, by the functional inactivity which it involved. His facts support the anatomical conclusions already quoted. The extension he gives to the region of cerebral vision in which lesions produce hemianopsia, is a reaction against the doctrines of Séguin, Nothnagel, and Henschen. The delimitation of this region is still, however, far from certain; as also the relation of the different parts of the occipital lobe to the various retinal segments. Monakow emphasises the importance of the primary optic centres interposed upon the optic path, such as the external geniculate body (in which the majority of the visual fibres terminate and lose their individuality). This fact is too often overlooked in schemata, which are constructed as if the projection bundles radiating over the visual sphere were the direct prolongation of the optic fillets.

This has important bearings upon the doctrine of retinal projection (*supra*). The anatomical conclusion appears to be that not the retinal fibres, but the fibres of the external geni-

culate body, are projected upon the occipital lobe. Monakow disallows the direct localised projection of impressions from the *macula lutea* on the cortex, his theory being that the macular fibres are distributed to all parts of the external geniculate body, and thus transmit their impressions to every part of the cortical centre. This would account for the partial preservation of central, or macular, vision in most cases of cortical hemianopsia.

Monakow's theory of the reciprocal relations, functional and trophic, that obtain between primary optic centres and occipital cortex is based upon secondary ascending and descending degenerations. These differ, histologically, and in their general physiological effects, according as the visual lesion is central or peripheral. After enucleation of the eye-ball in the dog, the ascending degenerations are sclerosis of the optic nerve, almost complete degeneration of the optic tract, slight diminution of volume in the motor nerve to muscles of the eye (oculo-motor, pathetic, external oculo-motor), general reduction of number of nerve cells, especially in the nucleus of the common oculo-motor. In dogs, cats, and man, the anterior quadrigeminal tubercles are little degenerated, while the external geniculate bodies are considerably atrophied—showing that most of the retinal fibres are closely connected with this body.

The same ascending atrophy was noted by Tomaschewski in the case of a child who became blind and deaf at the age of two, in consequence of meningitis. The ocular bulb atrophied from inflammation, and when the child died at the age of eight, the optic nerves, chiasma, tract, quadrigeminal tubercles, occipital lobe, angular gyrus, &c., were all atrophied.

Retrograde Degeneration.

Along with Wallerian degeneration, which attacks the peripheral end of a nerve separated from its trophic centre, there exists a retrograde degeneration, a "cellulipetal" alteration of the central end, propagated from the point of section to the trophic centre, *i.e.*, mother-cell of the nerve; and even in certain cases passing this centre to invade secondarily the following neuron. Marinesco (27) has observed a degeneration, varying in degree, in the central end of the peripheral nerves of amputated limbs. This involves the myeline and axis-cylinder, and determines in places a complete atrophy of the nerves, with proliferation of the interstitial tissue. Anatomically there is no essential difference between this process and that of Wallerian

degeneration at the peripheral end: it appears later, and advances more slowly; it is more rapid in young children than in adults.

Why do the nerves of the stump, and even cells in the anterior cornu, degenerate after amputation, seeing they are still in relation with their trophic centres, the spinal ganglia? The trophic centres themselves must suffer from the lesions of their nervous processes; the protoplasmic and axis-cylinder prolongations of a nerve-cell, with their terminal arborisations and collateral branches, are as much part of the cell as the limbs are part of the body of an individual; unity of the cell and its axis-cylinder is complete—a neuron is an individual. Golgi, Forel, Monakow, v. Gehuchten agree that lesion to any part of the neuron must bring about, first, the rapid destruction of that part of the nerve; and then the slower, but inevitable, death of the central portion of the neuron. The inter-annular segments of the nerves (“adipose cells traversed by the axis-cylinder of the nerve cell,” Durante) may constitute proper sources of nutrition, rendering the nerve, in some measure, independent of the parent cell. We do not, indeed, know whether the nerve derives its sustenance directly from the cell, or whether it borrows from local exchanges along its course, the nerve cell merely regulating the processes of assimilation. Undoubtedly, the nerve is subject to local influences from its environment, *e.g.*, effect of chemical agents in circulation. Renaut (Lyon) strongly urges the local reaction of a nerve independent of the ganglion cell. At the same time, the axis-cylinder must always be regarded as an integral part of the parent cell.

Marinesco refers the trophic functions of the nerve-cell, and their ability to nourish and preserve the structure of the neuron, to the continuity of peripheral excitations (thus reconciling functional inactivity and trophic influence in the neuron; Türk, Bouchard). The effects of interference of nutrition after amputation may extend to the cells of the anterior cornu, and thence to the motor nerves and the muscles supplied by them (*atrophy musculaire dégénérative*). Cerebral muscular atrophy may also be referred to interruption of functional excitation, and the hypothesis of a special cerebral trophic centre eliminated.

Retrograde degeneration is found along the sensory path of the optic nerve, as in the spinal nerves. Lesions of the occipital lobe, or external geniculate body, induce retrograde or cellulipetal degeneration in the neurons of the optic path, which may extend

as far as the tract and optic nerve, although each such neuron has its trophic centre in its own mother-cell. The external geniculate body and pulvinar degenerate after lesion of the visual area, *i.e.*, have no autonomous visual activity, or reflex activity—such as attaches to the optic lobes of birds; and Monakow believes that each segment of the thalamus is in exact functional and trophic relation with its own section of the cortex. The characteristics of cellulipetal degeneration are a more or less pronounced atrophy of the myeline, followed by its total disappearance, which leaves the axis-cylinders bare, till they, too, perish. Turning to clinical evidence, Moeli has shown that in adults, affections of the occipital lobe determine descending lesions that may be followed into the nerve cells of the retina. He cites two cases of foetal degeneration (porencephalia, internal hydrocephalia), due to lesion of the occipital lobe; the degeneration extended beyond the chiasma to the optic nerves. So, too, in another case: atheromatous softening of both occipital lobes in a man of 44, induced retrograde atrophy of the visual fibres and sub-cortical ganglia down to the optic nerve. Wilbrand, Henschen, and others found similar evidence for secondary descending degeneration. Nor does this stop at the optic nerve. The trophic disturbance of the basal ganglia affects the terminal arborisations of the opticus, and the descending alteration proceeds to the ganglion cells of the deep layers of the retina, and thence even to the rods and cones—the cellular prolongations in all cases being first to disappear.

M. Soury concludes from all these facts relating to the cortical localisation of bilateral, homonymous hemianopsia, that Ferrier is mistaken, and Munk correct, in their respective views concerning the cerebral centre of vision. Munk attributes the transitory hemianopsia, consequent on the extirpation of the angular gyrus, to the inflammatory reaction of the occipital lobe, and more especially to the compression (not destructive lesion) of the optic radiations of Gratiolet and Wernicke, which pass under the angular gyrus and inferior parietal lobe on their way to the cortex from the external geniculate bodies. It is thus that all lesions of the angular gyrus, &c., usually reach the projection bundle of the visual fibres. Should this be destroyed, and the message thus interrupted from the last sub-cortical station to the occipital cortex, nothing can avert absolute cortical, hemianopic blindness in the two halves of the retinae implicated. Séguin (38), Forel, Nothnagel concur in locating the seat of vision, the optic field of perception, in the cuneus and first occipital gyrus. Unilateral lesion of these parts

produces hemianopsia, bilateral lesion produces total blindness.

HEMIOPIC HOMONYMOUS HALLUCINATIONS.

Tamburini defines hallucinations as a kind of epilepsy of the sensory centres. Visual hallucinations in the obliterated half of the field of vision may often be observed in hemianopsia of cortical origin. This is an extra phenomenon, super-added on the destructive lesion that produces partial cecity in both eyes, and due to an irritative lesion of the occipital cortex. Irritative processes resulting in hallucinations are frequently the precursor of destructive occipital lesions. Such visual hallucinations may even serve to determine the precise point of the cerebral cortex that is the seat of the lesion, *i.e.*, the hallucinatory alterations in the field of vision may be projected back upon the corresponding region of the "cortical retina." Soury does not accept the restriction of this irritative lesion to the calcarine surface of the occipital lobe (Lamy, *cf.*). He holds that the associations adequate to realise complex hallucinations must extend over much wider regions, probably over the convexity of the occipital lobe. He queries the possibility of unilateral hallucinations, and holds that just as a unilateral destructive lesion of the occipital lobe determines a homonymous bilateral hemianopsia, which produces partial cecity in both corresponding halves of the visual field, so a unilateral irritative lesion of the same region must determine a bilateral homonymous hallucination, which partially affects the visual fields of both eyes. Clinical evidence is quoted from Peterson, Colman, Hun, and Pick.

F. A. WELBY.

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(1) This is a *brochure* of about 140 pages, giving in a readable form the work of R. y Cajal upon the minute anatomy of the medulla oblongata, the pons Varolii, cerebellum, and origin of the cranial nerves. There is little to note that is not by now well known to most workers upon the anatomy and physiology of this region—facts which are gradually finding their way into the text-books, and which have already done much to explain many of the curious phenomena brought out by disease. The book is well illustrated, and there is an ample bibliography at the end.

(2) This is the record of an experimental research, performed with a view to trace the several bundles of fibres which constitute the optic nerve, optic chiasma and optic tract. A wound was made in the retina by means of a galvano-caustic needle, the operator being guided by the aid of the ophthalmoscope. After a limited time the animals were killed, and the nerves examined by the Marchi method. The following is a summary of the results: The fibres maintain the same relative position throughout the optic nerve and tract. In the chiasma, the decussation takes place in a regular order, the innermost bundles of the nerve crossing first, and occupying an outer position in the tract, the lower bundles of the nerve occupying the lower part of the tract, and the outer bundles occupying the inner part.

It is probable (although there was a technical difficulty in the operation) that the uppermost bundles occupy the upper part of the tract. The decussation of the fibres is only in a transverse direction, there being no alteration of the fibres in a vertical direction. The several segments of the nerve correspond in general to like segments of the retina.

W. A. TURNER.

BRAIN.

PART IV., 1896.

Original Articles and Clinical Cases.

ON THE EVOLUTION OF HYPNOTIC THEORY.

BY DR. J. MILNE BRAMWELL.

IN attempting to present a picture of hypnotic theories the first difficulty encountered is due to their number and diversity. Max Dessoir, in his "Bibliography of Modern Hypnotism," published in 1888, cites 800 works by nearly 500 authors, and it would be difficult to find two of them agreeing in every detail as to the theoretical explanation of all hypnotic phenomena.

Apparently little of value has been discovered which can justly be considered as supplementary to Braid's later work, while much has been lost through ignorance of his researches. In the successful exposure of the errors of the Charcot School by Bernheim and his colleagues, is to be found a reproduction of Braid's controversy with the mesmerists; while the Nancy theories themselves are only an imperfect reproduction of Braid's later ones.

Braid's Theories.

According to Braid's earlier or physical theory the explanation of hypnotic phenomena was to be found in changes in the system of the subject, not in that of the operator. These consisted in the exhaustion of certain nerve centres, with

resulting decrease in the functional activity of the central nervous system, and arose from continued monotonous stimulation of other nerves by means of fixed gazing, passes with contact, and similar methods. Braid experimentally proved that the phenomena supposed to result from mesmeric or odyllic force only appeared when the various physical stimuli were associated with mental impressions, and were invariably absent when these were excluded.

At a later date Braid adopted a more purely psychical explanation of hypnotic phenomena. To this I will again refer, meanwhile I wish to call attention to other physical theories, pathological as well as physiological.

Charcot's Theory or that of the Salpêtrière School.

In the opinion of this School, hypnosis is an artificially-induced morbid condition, a neurosis only to be found in the hysterical. Women are more easily influenced than men, children and old people are almost entirely insusceptible. Hypnosis can be produced by purely physical means; and a person can be hypnotised, as it were, unknown to himself. Hypnotic phenomena are divided into three stages, which are induced and terminated by definite physical stimuli. Hypnotism, so far, has not proved of much therapeutic value. There is danger of provoking hysteria in trying to induce hypnosis. Certain hypnotic phenomena can be induced, terminated, or transferred by means of magnets, metals, &c. There does not exist a single case in which a somnambule has acted criminally under the influence of suggestion. There is a difference between suggestion in normal life, and in hypnosis. The former is a physiological phenomenon, the latter a pathological one. Suggestibility does not constitute hypnosis, it is only one of its symptoms.

This theory has been strongly attacked, chiefly by the so-called Nancy School. To commence with, they point out the insufficiency of the data upon which it has been founded, and cite the confession of one of its supporters that only a dozen cases of true hypnosis have occurred in the Sal-

pêtrière in ten years, and that a very large proportion of the experiments were conducted with one subject, who had long been an inmate of that hospital. On the other hand, they call attention to the extended nature of their own observations, and to the fact that their conclusions are drawn from the study of many thousand cases.

Is Hypnosis a Morbid Condition which can only be Induced in the Hysterical?

This question must, I think, be answered in the negative. Charcot argued that hypnotism and hysteria were identical, because in both the urine presented similar characteristics. In reply to this, Moll pointed out that all Charcot's subjects suffered from hysteria, and, as the phenomena which characterise waking life are readily induced in hypnosis, Charcot easily created a complete type of hysteria by suggestion.

If the hysterical alone can be hypnotised, we must conclude from the following statistics that at least 90 per cent. of mankind suffer from hysteria. Some years ago Bernheim had already attempted to hypnotise 10,000 hospital patients, with over 90 per cent. of successes. In 1893, Wetterstrand, of Stockholm, reported 6,500 cases, of which 105 or 3·7 per cent. remained uninfluenced. Liébeault informed me that he had no complete record of the thousands of cases he had treated in over thirty years' practice, but that from 1887 to 1890 he had tried to hypnotise 1,756 persons with a fraction over 3 per cent. of failures. Van Eeden and van Renterghem, of Amsterdam, from 1887 to 1893, report 1,081 cases, with 5·33 per cent. of failures.

Mr. Hugh Wingfield, when demonstrator of physiology at Cambridge, hypnotised 152 undergraduates at the first attempt. Most of them would be drawn from our public schools, and if these do not always turn out good scholars they cannot, at all events, be accused of producing hysterical invalids. Liébeault found soldiers and sailors particularly easy to influence, while Grossmann, of Berlin, recently asserted that hard-headed North Germans were very sus-

ceptible. Professor Forel, of Zürich, told me he had hypnotised nearly all his asylum warders: that he selected these himself, and certainly did not choose them from the ranks of the hysterical. Braid also found that the nervous and hysterical were the most difficult to hypnotise. When Esdaile, who might justly be called the father of anæsthetic surgery, was performing painless operations before the introduction of ether and chloroform into India, his results were ascribed to the influence of hysteria. He recorded 261 painless capital operations and many thousand minor ones, and reduced the mortality in the removal of the enormous tumours of elephantiasis from 50 to 5 per cent. His patients were nearly all males, and he remarked: "I cannot possibly see how hysteria can have got into my hospitals, where I never saw it before, coolies and felons not being at all nervous subjects."

These and similar facts apparently justify the statements of Forel and Moll that it is not the healthy but the hysterical who are the most difficult to influence. The former considers that every mentally healthy man is naturally hypnotisable; while the latter says, if we take a pathological condition of the organism as necessary for hypnosis, we shall be obliged to conclude that nearly everybody is not quite right in the head. The mentally unsound, particularly idiots, are much more difficult to hypnotise than the healthy. Intelligent people, and those with strong wills, are more easily hypnotisable than the dull, the stupid, or the weak-willed. Forel says the most difficult to influence are without doubt the insane; while the number of mentally healthy persons hypnotised by Liébeault and Bernheim alone amounts to many thousands. My personal experience accords with these views. I formerly found no difficulty, for example, in hypnotising healthy Yorkshire peasants for operative purposes, and amongst that class obtained 100 per cent. of successes. Now, when my patients are usually chronic nervous invalids, I find the difficulty of inducing hypnosis greatly increased. This experience is not alone a personal one; for, on visiting hypnotic clinics in France, Switzerland, Holland and Sweden, I invariably found that others encountered similar difficulties.

Are Women more Susceptible than Men?

All observers, with the exception of the Salpêtrière School, agree in stating that sex has little or no influence upon susceptibility. According to Liébeault, the difference between the two sexes is rather less than one per cent. All Wingfield's, and the majority of Esdaile's, subjects were men.

Are Children and Old People Insusceptible?

Wetterstrand found that all children from 3 or 4 to 15 years of age could be influenced without exception. Bérillon, out of 250 cases in children, hypnotised 80 per cent. at the first attempt. Liébeault also found them peculiarly susceptible; and one of his statistical tables records 100 per cent. of successes up to the age of 14. In adult life, age apparently makes little difference. In the same table we find that from the ages of 14 to 21 the failures were about 10 per cent., and from 63 years and upwards about 13 per cent.

Can Hypnosis be induced by Mechanical Means alone?

This question is answered by the Nancy School in the negative, and my own experience agrees with this. I know of no single instance where hypnosis has followed the employment of mechanical means, when mental influences have been carefully excluded, and the subjects have been absolutely ignorant of what was expected of them. No one was ever hypnotised by looking at a lark-mirror, until Luys borrowed this lure from the bird-catchers, and invested it with hypnotic power. On the other hand, any physical method will succeed with a susceptible subject who knows what is expected of him.

Are Hypnotic Phenomena divided into Three Distinct Stages?

The stages described by the Salpêtrière School as arising from definite physical stimuli, have never been noticed by other observers. Amongst the many hundred hypnotised

subjects I have seen, none have responded to the manipulations which produced such striking phenomena at the Salpêtrière. On the other hand, I and many others have found that we could easily evoke these stages by verbal suggestion, and train the patients to manifest them at a given signal. The condition, however, was always an artificial one.

Is Hypnotism of little Therapeutic Value?

On the one hand, we have the negative evidence of a few cases observed at the Salpêtrière, where experiment, not cure, seemed the main end. On the other, we have the positive evidence drawn from many thousands of cases, where hypnotism has been successfully employed for the relief or cure of disease.

According to Professor Benedikt, of Vienna, however, the majority of the results attributed to hypnotic suggestion are fallacious, the patient simply deceiving the physician. As to the alleged cures of alcoholism, he asks, where are these cases? There can be no possible doubt, he says, that the medical men who reported them are to-day aware of their error. Why are they silent? The true explanation of the supposed cures is simply to be found in the fact that the patients want to be left alone, and therefore say they are cured. Amongst so-called successful cases at least 90 per cent. must be eliminated for this reason.

This opinion is entirely opposed to facts. Those who formerly reported cases of hypnotic cure continue to do so, while many others have joined their ranks. The following, amongst others, may be quoted as having recently testified to the therapeutic value of hypnotic treatment: van Eeden, van Renterghem, Forel, Freud, Gerster, Grossmann, de Jong, Scholz, von Schrenck-Notzing, Tatzel, Wetterstrand, Brunnberg, Hecker, Krafft-Ebing, Ringier, Bergmann, Brügelmann, Fulda, Herzberg, Hirt, Schmidt, Vogt, Schmeltz, Lemoine, Joire, Voisin, de Mézeray, Bérillon, Bernheim, Liébeault, Dumontpallier, Gorodichze, Bonjour, Desplats, Bourdon, Tissié, &c.

Benedikt would like to know what has become of the dipsomaniacs said to have been cured by hypnotism. As regards my own cases, I can give a very satisfactory reply. Some are actively engaged in business, or in successfully conducting medical practice; one has since been elected a Member of Parliament, while others are happy wives and mothers. In most of them the disease had been of long duration, varying from about five to fifteen years; and in some presented all its worst symptoms; for example, the patient who is now a Member of Parliament had formerly several attacks of delirium tremens and epilepsy. The duration of the cure has lasted from two to over six years.

Benedikt's suggestion that dipsomaniacs pretend to be cured in order to be left alone is amusing. Drunkards sometimes secretly obtain drink in ingenious ways, but I never heard of one being able successfully to pretend that he was sober when dead drunk, or in good health when suffering from delirium tremens.

In the "*Zeitschrift für Hypnotismus*," part 1, 1896, Wetterstrand reported 38 cases of morphinism treated by hypnotic suggestion. Of these 28 were cured, 3 relapsed, and in 7 no result was obtained. In each instance, the morphia had been injected subcutaneously. Many of the cases were exceedingly grave and of long standing, and some were complicated with the cocaine and alcohol habit. With several the abstinence treatment had been tried without success—sometimes more than once. One of the successful cases—a medical man—had taken morphia for eighteen years, and during the last four years cocaine also. Another medical man, Dr. Landgren, recorded his own case in the same journal. Over five years have elapsed since he was successfully treated by Wetterstrand. Other methods, including residence in a retreat, &c., had failed.

I do not for a moment pretend that by hypnotism one can cure everything and everybody, and agree with Braid in thinking that he who talks of a universal remedy is either a knave or a fool. On the other hand, I have seen many cases of functional nervous disorder cured or relieved by hypnotism, which had previously resisted long careful

and varied medical treatment. For example, I recently reported eight cases of "Imperative Ideas," in which hypnotic suggestion had been successfully employed, the first dating from 1889, the last from 1894. Of these one afterwards died of influenza, but the others remain well. In some instances the original mental trouble had been a grave one, and the patients had suffered from delusions and hallucinations.

Is Hypnotism Dangerous ?

The Salpêtrière School answer this in the affirmative, asserting that hysterical symptoms have sometimes appeared after the attempted induction of hypnosis. That such phenomena should occur with them is not surprising, when one considers the nature of the patients and their surroundings, and the violent and startling methods sometimes resorted to. The slight accidents which they record have not occurred in other and more experienced hands. Professor Forel says : " Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, Moll, I myself, and the other followers of the Nancy School, declare categorically that although we have seen many thousands of hypnotised persons, we have never observed a single case of mental or bodily harm caused by hypnosis ; but, on the contrary, have seen many cases of illness relieved or cured by it." This statement I can fully endorse, as I have never seen an unpleasant symptom, even of the most trivial nature, follow the skilled induction of hypnosis.

In the *Revue Médicale de l'Est*, February 1, 1895, Bernheim records the only case, as far as I know, in which death followed hypnosis induced by a medical man. The patient suffered from phlebitis, accompanied by severe pain ; and to relieve this, Bernheim hypnotised him. He died two hours afterwards, and *post-mortem* examination showed that death was due to embolism of the pulmonary artery. The case is referred to in the *British Medical Journal*, and though it is admitted that the occurrence was nothing more than an "unlucky coincidence," it is stated, at the

same time, that "it is at least arguable that the psychical excitement induced by the hypnotising process may have caused a disturbance in the circulatory system, which had a share in bringing about the catastrophe." Bernheim has hypnotised over 10,000 hospital patients; sometimes this would be done for the relief of pain associated with inevitably fatal maladies, and therefore, the matter for surprise is that death has not frequently occurred during, or shortly after, the induction of hypnosis. The majority of fatal illnesses receive medical treatment: it would then, according to the theory of the *British Medical Journal*, be justifiable to argue that the administration of drugs "may have had a share in bringing about the catastrophe." Certainly their use is likely to be attended with more physical and psychical excitement than is involved in the hypnotising processes in vogue at Nancy.

Such arguments against hypnotism are dangerous and apt to provoke unpleasant replies. For example, Moll, in reference to some hostile criticisms of Ziemssen, said: "If Ziemssen had shown the same scepticism when the tuberculine craze excited all Germany, much injury to science and to his patients would have been prevented. The wantonness with which at that time the lives of many were staked will remain as a lasting blemish upon science; and it cannot be denied that the excessive use of tuberculine was the cause of the untimely death of many human beings. In ordinary life, one would describe such a proceeding as an offence against the person, of which the issue was death. I cannot recognise that there is a peculiar law for clinical professors; and that when they have, in such a manner, hastened the death of a human being, another expression should be used."

Can various Physical and Mental Phenomena be excited by the Application, or near Presence of, certain Metals, Magnets, and other Inanimate Objects?

Here, in the assertions of the Salpêtrière School, and their refutation by that of Nancy, we have an exact counterpart of the controversy between Braid and the mesmerists. All

the old errors, the result of ignoring mental influences, are once more revived. Medicines are again alleged to exercise an influence from within sealed tubes. The physical and mental conditions of one subject are stated to be transferable to another, or even to an inanimate object. It is useless to enter into any arguments to refute these statements, for this would be needlessly repeating the work of Braid. Indeed, in many instances, their absurdity renders argument unnecessary. For example, when a sealed tube, containing laurel-flower water, was brought near a Jewish prostitute, she adored the Virgin Mary! From this it might be inferred that different religious beliefs were represented by different nerve centres, and that these could be called into action by appropriate physical stimuli. Should this be established, it could hardly fail to have an important influence upon the character and direction of missionary enterprise! The chief apostle of these doctrines is Luys; and considerable attention was drawn to them in this country in 1893 by popular articles in the daily papers and elsewhere. Indeed, the editor of a well-known medical journal thought them of sufficient importance to demand his writing a book in order to disprove them. He apparently was ignorant of the fact that M. Dujardin-Beaumetz had, in 1888, reported to the Académie de Médecine that Luys' experiments were conducted so carelessly as to rob them of all value, and that among students of hypnotism they are entirely disregarded.

While Luys believes that magnets, &c., excite extraordinary hypnotic phenomena, we find, strange to say, Benedikt expressing somewhat similar views as to their influence on non-hypnotised subjects. According to Benedikt, certain forms of hysteria are better treated by the magnet than by electricity, hydropathy, or drugs. He continues as follows: when the magnet is applied to the sensitive vertebræ, without removal of the dress, the irritable patient soon becomes quiet, and even quasi-paralysed. The muscles gradually relax, the respiration becomes sighing, consciousness slowly disappears. The resistance to conduction in motor nerves would easily become absolute. The two poles

have different effects. The magnet must be employed with caution: the patients may be injured by it. These statements were recently tested at the Edison Laboratory, in America; magnets of enormous power were used,¹ and experiments made on human subjects and lower animals. A young dog was subjected to magnetic influence for five hours; but, instead of being paralysed from increased resistance to conduction in motor nerves, on being liberated it was more lively than before. The experimenters conclude that the human organism is in no wise appreciably affected by the most powerful magnet known to modern science; that neither direct nor reversed magnetism exerts any perceptible influence upon the iron contained in the blood, upon the circulation, upon ciliary or protoplasmic movements, upon sensory or motor nerves, or upon the brain. The ordinary magnets used in medicine, they say, have a purely suggestive or psychic effect; and would, in all probability, be quite as useful if made of wood.

The two remaining points, namely, the question of criminal suggestion and that of suggestion in general in relation to hypnotism, I will refer to in discussing the theories of the Nancy School.

Heidenhain's Theory.

According to Heidenhain, the phenomena of hypnotism owe their origin to arrested activity of the ganglion-cells of the cerebral cortex. He holds that these higher centres are inhibited by the monotonous stimulation of other nerves, *i.e.*, by fixed gazing, passes, &c., and that sensory impressions, which usually produce movements after passing to the higher centres and evoking consciousness, now do so by passing directly to the motor centres. This is essentially a "short-circuiting of nervous currents" theory. Heidenhain regards the hypnotised subject as a pure automaton, who imitates movements made before him, but who is entirely unconscious of what he is doing. To cause him to move his arm, he says, the image of a moving arm must pass before his retina, or an unconscious sensation of

¹ Electro-magnets of 2,000 to 5,000 C.G.S. units to the square centimetre were employed.

motion must be induced through passive movement of his arm. The patient has no idea corresponding to the movements he makes ; the sensory impression leads to no conscious perception and to no voluntary movement, but suffices to set up unconscious imitation. In reference to the playing of different parts by hypnotised subjects, Heidenhain says that it is a mistake to suppose that the subjects realise what they are doing : this is quite out of the question ; the hypnotised individual neither thinks nor knows anything about himself. Heidenhain holds that the fact of the hypnotised subject's forgetfulness of the sensations he has experienced during hypnosis affords satisfactory evidence that these sensations were unconscious ones. This theory was first published in 1880, and attracted considerable attention. It was accepted, for example, by Professor McKendrick, of Glasgow, and restated by him in the last edition of the "Encyclopædia Britannica," as giving a true and scientific explanation of the phenomena of so-called animal magnetism.

To this explanation many objections may be urged, thus :—

(1) It is a mistake, as has been pointed out, to call the hypnotic action on the cortical functions *inhibition*, without stating explicitly that the normal action of these functions in respect to motion is always to a large extent inhibitory, and that the complete description of the method by which the so-called automatic reflex responses are brought about is thus "inhibition of the inhibitory functions."

(2) While giving an elaborate exposition of the theory of cerebral *inhibition* produced by peripheral stimulation, Heidenhain omits to take into consideration the result of central *stimulation* by means of an idea or emotion. As hypnosis can be equally well induced in this way, we cannot accept Heidenhain's theory, as he wishes that we should do, in substitution for that of "dominant ideas." It cannot justly be considered as an alternative to it, as it is simply the physiological statement of psychical facts.

(3) The theory itself is not a new one ; with the exception of a few details, it is essentially an imperfect reproduc-

tion of that of the late Professor John Hughes Bennett, of Edinburgh, published in 1851. But Bennett, as we shall see later, possessed a clearer view of the whole problem, and did not fall into the mistake of attempting to substitute the physiological statement of psychical facts for the facts themselves.

(4) Hypnosis can be induced, not only in the absence of *monotonous* peripheral stimulation, but even without any peripheral stimulation at all. At the present day it is usually evoked by central stimulation, which, in those who have been previously hypnotised, need not be persistently monotonous; the single word "sleep" being then sufficient to excite the condition. In such cases, the factor which Heidenhain regards as essential to the production of hypnotic phenomena, *i.e.*, monotonous peripheral stimulation, is absent.

(5) Instead of a hypnotised subject imitating a movement which he sees, and failing to perform one which is verbally suggested to him, the reverse is actually the case. As a rule the slightest verbal suggestion is sufficient to induce the movements described by Heidenhain; on the other hand, hypnotised subjects never copy movements made before them unless they have been trained to do so. The imitative movements only take place, according to Moll, when the hypnotic subject is conscious of them, and knows that he is intended to make them. If they were unconscious reflexes, the subjects would imitate any person's movements, but they only imitate the one person who exists for them, *i.e.*, the experimenter, and him only when they know he wishes them to do so. When such experiments are often repeated the imitation may become automatic in later hypnoses, as happens in waking life. At first, however, a clear idea of the movements to be made is necessary; but as we regard the cerebral cortex as the seat of ideas, and as there is no reason for shifting them to another part of the brain in hypnosis, so there can be no doubt of the activity of the cerebral cortex.

(6) Heidenhain's only argument is based upon the subject's subsequent defect of memory; he thus assumes, as his

sole test for present consciousness, the subsequent remembrance of its content. Yet, as has been pointed out, if the reality of that test be granted, the question whether a man was conscious when he read an article in the *Times*, will depend on whether or not he received a blow on the head when he had finished it. There is a more radical objection to all these arguments founded on subsequent loss of memory, the fact, namely, that memory is frequently present. Braid, for example, found that only some 10 per cent. of his subjects were unable to recall the events of hypnosis, while Schrenck-Notzing's International Statistics give 15 per cent. in 8,705 cases. Again, if on waking, the act performed during hypnosis is forgotten, the lost memory can be revived in subsequent hypnoses; and, finally, the amnesia, which would otherwise follow deep hypnosis, can be entirely prevented by suggestion.

Mr. Ernest Hart's Theories.

Mr. Hart has also attempted to explain the phenomena of hypnotism from a physical standpoint. According to him, the hypnotised subject is reduced more or less perfectly to the state of a living automaton; the upper brain is more or less completely, and more or less regularly, bloodless, its functions are in abeyance, the will is abolished, suspended or enfeebled. A hypnotised subject, he says, will perform unconsciously, under the influence of suggestion, acts which are dangerous to himself and others, and which are in themselves criminal—so that he can be made to thief, to commit arson, or to attempt violence. He summarises his views in the following somewhat obscure sentence: "We have already, I hope, succeeded in eliminating from our minds the false theory—the theory that is experimentally proved to be false—that the will or the gestures, or the magnetic or vital fluid of the operator is at all necessary for the abolition of the consciousness and the abeyance of the will of the subject; and we now see that ideas arising in the mind of the *subject* are sufficient to influence the circulation in the brain of the *person operated on*, and in such

variations are adequate to produce sleep in the natural state, or artificially by total deprivation, or by excessive increase, or local aberration in the quantity or quality of the blood to produce coma and prolonged insensibility by pressure of the thumbs upon the carotid; or hallucinations, dreams, and visions by drugs, or by external stimulation of the nerves, or to leave the consciousness partially affected, and the person in whom sleep, coma, or hallucinations is produced, subject to the will of others and incapable of exercising his own."

Mr. Hart agrees with the Charcot School as to the connection between hypnotism and hysteria, and says: "I fully admit that under the influence of certain psychological stimuli, persons whose nervous system is ill-balanced, or at best in a condition of unstable equilibrium, readily pass into a state of hypnotic sleep." In discussing this question I have already attempted to show the erroneous nature of these and similar views. There remain, however, three other objections to Mr. Hart's theories.

(1) It is doubtful whether any of the phenomena—unconsciousness, automatism, susceptibility to criminal suggestion—which he regards as characteristic of the hypnotic state, exist. The general question of consciousness and automatism has already been referred to in dealing with Heidenhain's theory; that of criminal suggestion I propose to discuss more fully in connection with the views of Bernheim and others. Meanwhile, I may state, as the result of seven years' hypnotic research, that I have failed, either at home or abroad, to discover a single instance in which the phenomena described by Mr. Hart have occurred.

(2) The presence of cerebral anæmia in hypnosis is by no means established. The belief in its existence is an old one, which recent investigations have done much to discredit. Many years ago Carpenter suggested it as a possible explanation of at least some hypnotic phenomena, and Hack Tuke also considered there was a partial spasm of the cerebral blood vessels in hypnosis. Heidenhain, too, at first supposed that anæmia of the brain was the cause of hypnosis. He soon gave up this opinion for several reasons. (*a*) He saw hypnosis appear in spite of the inhalation of nitrite of

amyl, which causes hyperæmia of the brain. (b) The investigations of Förster discovered no change in the vessels at the back of the eye during hypnosis. (c) Salviole and Bouchut stated that they found cerebral hyperæmia during hypnosis.

(3) Mental activity varies in hypnosis just as it does in normal life, and in both is doubtless associated with changes in the blood supply; but, even granting that cerebral anæmia exists in hypnosis, to assume that it explains its phenomena is unscientific. For, as Professor William James points out, the change in the circulation is the result, not the cause, of the altered activity of the nervous matter. Many popular writers, he says, talk as if it were the other way about, and as if mental activity were due to the afflux of blood; that belief has no physiological foundation whatever; it is even directly opposed to all that we know of cell life. The stomach does not digest because more blood flows into it, nor do the muscles of the arm contract for a similar reason; on the contrary, their increased blood supply follows their increased functional activity. If one desired to be hypercritical, one might still further object that when a correlation had been established between a physical condition and a mental state, the one did not in any true sense explain the other. As Tyndall said: "There is no fusion possible between the two classes of facts. The passage from the physics of the brain to the corresponding facts of consciousness is unthinkable."

Hypnosis in Animals.

Mr. Hart believes that animals, such as guinea-pigs, rabbits, frogs, birds, crayfish, and even young alligators can be hypnotised by methods similar to those employed with the human subject, and that they present like phenomena. The only argument in favour of this is drawn from the fact that these animals, after certain physical stimuli have been applied to them, present the phenomenon of catalepsy. Is this catalepsy invariably a genuine one? I am inclined to think that in many instances it is a conscious simulation of death, adopted by the animal from the instinctive knowledge of the

fact that certain birds and beasts of prey, except under pressure of extreme hunger, will not attack what is dead. If, for example, you turn a beetle over on its back it will remain motionless and apparently cataleptic, with its legs sticking rigidly in the air. The moment you go away, however, it scrambles to its feet and resumes its journey. Here death, or catalepsy, was in all probability only shammed, and, doubtless, the insect was keenly watching your every movement, and anxiously waiting for your departure. Again, catalepsy is only one, and a comparatively unimportant, phenomenon of hypnosis. One of the main characteristics of the hypnotic state is the rapidity with which one phenomenon can be changed into its opposite. Have we any evidence of this in the so-called hypnosis of animals? I think not. Again, is it logical to conclude similiarity of cause from identity of effect? In order to induce hypnotic catalepsy in the human subject, a clear idea of the suggested act is necessary.¹ What evidence have we for concluding that a crayfish becomes cataleptic from a clear idea that the operator has suggested this condition? It is possible that in some instances the phenomenon is genuine; but we find it explained in varying, and even opposite, ways by different observers. Thus, Heubel and Wundt consider the so-called hypnosis of animals a true sleep, resulting from the *cessation* of external stimuli. Preyer, on the contrary, believes the condition to be one of paralysis from fright, or catalepsy produced by sudden peripheral stimulus. Now fear is not necessary for the induction of hypnosis; and, before concluding that the condition is a hypnotic one, it would be wise to exclude this factor from the equation. To do this experimentally would not be difficult; it would only be necessary to get rid of the disproportion in size and strength which, in the experiments referred to, has always existed in favour of the hypnotiser. Instead of a young alligator, let one of greater age and larger growth be chosen, and the experi-

¹ Verworn has recently shown that this condition in animals is one of tonic contraction of the muscles, and is determined by an excitation of the nervous elements of the mid-brain. It can be induced in fowls deprived of their cerebral hemispheres, while in the human subject a clear idea of the suggested act is necessary.

ment repeated. I am inclined to think that in such a case the rôles would be reversed, the operator would become cataleptic, and the subject uncommonly and disagreeably mobile !

Again, Mr. Hart holds that hypnosis can be induced in the hysterical alone, in those possessing ill-balanced nervous systems. What evidence have we for the existence of hysteria in the alligator? I think none ; and yet the spectacle of an alligator executing *grands mouvements*, or even *mouvements passionnels*, would have been certain to have attracted attention.

The Contradictory Nature of Hypnotic Theories.

Moll draws attention to the fact that these physiological and pathological theories frequently contradict each other. Thus, Rieger and Conrad regard hypnosis as an artificial mania, while Meynert maintains that it is an experimentally produced imbecility : two forms of mental disease which are utterly dissimilar. Semal and Hack Tuke also called hypnosis an artificially induced insanity. We might, says Moll, call hypnosis an insane condition, if we also regarded sleep and dreams as such. When psychologists wish to discover analogies to mental disorders, they always have recourse to dreams ; but no one maintains that in order to lose our sanity it is only necessary to go to sleep. Two conspicuous characteristics of hypnosis are suggestibility and the fact that the state can be terminated at will ; but we do not find these united in mental disorders, or in neuroses. While physiologists fail to consider what an enormous influence an idea, aroused, for example, by the word "wake," exercises, their theories will ever remain unsatisfactory. We ought, Moll continues, to set our faces decidedly against the way in which certain physiologists juggle with words, as if the enigmas of consciousness were child's play to them. When Mendel, speaking of the phenomena of hypnosis, explains that we have to do with a strong stimulation of the cerebral cortex and Ziemssen declares the exact contrary, *i.e.*, that the cerebral cortex is too little stimulated, and the sub-cortical centres too much, we are startled at such contradictions, and can only hope that in the future less will be asserted and more proved.

Braid's Later or Psychological Theory.

According to this theory, the hypnotic condition was essentially one of mental concentration or monoideism, in which the mind was so engrossed with a single idea as to render it dead to all other influences. All the phenomena, no matter how hypnosis was induced, resulted entirely from *dominant ideas* aroused in the minds of the subjects.

The attention was concentrated upon the particular function called into action, while the others passed into a state of torpor. Only one function was active at any one time, and hence intensely so. The arousing of any dormant function was equivalent to superseding the one in action.

In his earlier theories, Braid attached most importance to the physiological changes associated with hypnosis; in his later, to the psychological ones. He never entirely separated the two, however; and when he explained the condition by means of monoideism, he still clearly stated that this depended upon a definite physical change in the subject. This resulted from the methods employed to induce hypnosis, and alone rendered the mental condition possible. In his later works he gives no further elaboration of his physical theory, and for this we must turn to Professor John Hughes Bennett, who adopted Braid's view in reference to monoideism, and explained it both from the physiological and psychological side.

Bennett's Physiological Theory.

According to Bennett, hypnosis is characterised by alterations in the functional activity of the nerve tubes of the white matter of the cerebral lobes. He suggested that a certain proportion of these became paralysed through continued monotonous stimulation; while the action of others was consequently exalted. As these tubes connect the cerebral ganglion cells, suspension of their functions was assumed to bring with it interruption of the connection between the ganglion-cells.

Bennett's Psychical Theory.

" From the psychical side, he explained the phenomena of hypnosis by the action of predominant and unchecked ideas. These were able to obtain prominence from the fact that other ideas, which, under ordinary circumstances, would have controlled their development, did not arise; because the portion of the brain with which they were associated had its action temporarily suspended, *i.e.*, the connection between the ganglion-cells was broken, owing to the interrupted connection between the fibres of association. Thus, he says, the remembrance of a sensation can always be called up by the brain; but, under ordinary circumstances, from the exercise of judgment, comparison, and other mental faculties, we know it is only a remembrance. When these faculties are exhausted, the suggested idea predominates, and the individual believes in its reality. In this manner we attribute to the faculties of the mind, as a whole, a certain power of correcting the fallacies which each one of them is liable to fall into; in the same way that the illusions of one sense are capable of being detected by the healthy use of other senses. There are illusions mental and sensorial: the former caused by predominant ideas, and corrected by proper reasoning; the latter caused by perversion of one sense, and corrected by the right application of the others.

In hypnosis, then, according to this theory, a suggested idea obtained prominence and created mental and sensorial illusions; because the check action—the inhibitory power—of certain higher centres had been temporarily suspended. These theories were first published by Professor Bennett in 1851.

If we confine ourselves entirely to the psychological side of this conception of hypnosis, we find an almost identical picture of the condition presented by certain later observers, such as Edmund Gurney and Bernheim.

Gurney's Theory.

After referring to the certain and isolated way in which suggestion evokes a particular idea in the mind of the sub-

ject, Gurney states that this isolation of a single object in the mind naturally implies abeyance of the normal controlling and relating powers. In the normal state, he says, successive vivid points of consciousness are surrounded by a swarm of subordinate perceptions and ideas, by reference to which conduct is instinctively or sub-consciously kept rational. In the hypnotic state the contact is broken between the predominant idea and this attendant swarm; and conduct thus ceases to have reference to anything except the predominant idea. The hypnotic mind is working with marked absence of individuality in the channel chosen by others; and what its owner says or does, in response to external influence, is that on which his attention is concentrated, to the complete exclusion of every other thought.

Bernheim's Theory.

In hypnosis, according to Bernheim, the whole nervous force of the subject is concentrated upon a single idea. This nervous concentration may be changed from one point to another in response to the suggestions of the operator; but, though the focus shifts its place, the same concentration continues to exist.

In the normal state, he says, we are subject to errors, illusions, and hallucinations. Sometimes these are spontaneous, or follow imperfect sensorial impressions; at others they are suggested to us, and accepted without being challenged. In the normal state there is a tendency to accept ideas suggested by others, and to act upon them; but every formulated idea is questioned, and, as the result of this, either accepted or rejected. In the hypnotised subject, on the other hand, there exists a peculiar aptitude for transforming the suggested idea into an act. This is so quickly accomplished that the intellectual inhibition has not time to prevent it; and, when it comes into play, it does so too late, as the idea has been translated into its physical equivalent. If consciousness follows the suggested act, it at all events follows it too late to interfere with its fulfilment.

While Bernheim almost exactly reproduces the views of

Braid and Bennett in reference to the general mental condition in hypnosis, he and certain other members of the Nancy School differ from them in many important points; and to these I now propose to call your attention.

But first a word in reference to the so-called School of Nancy, a term which Benedikt ridicules in apparent ignorance of what Professor Beaunis had previously written on the subject. Beaunis says: "We hear frequently nowadays of the School of Nancy, but there is something in this term which has been applied to us which does not correspond with the reality. The term, "School," implies a community of which all the members hold the same ideas. This we are not; but the public quickly accept ready-made labels without troubling themselves much about the ideas underneath them. In our researches, undertaken with mistrust and doubt, we have arrived at similar results upon a certain number of points. Each observer, however, retains his own ideas and individuality, and it is easy to note the profound and even radical differences which separate us. These diversities of opinion are easily explained, and their non-existence would be impossible in a science still in its infancy. What is common to all of us is the conviction of the importance of these questions, and the belief in their future—the profound conviction that this method so ridiculed constitutes one of the greatest advances of the human mind and one of its most precious possessions."

Points of Difference.

Braid and Bennett, as we have seen, regarded the phenomena of hypnosis as the result of a definite physical change in the subject; Bernheim, on the other hand, attempts to explain them (1) by finding an analogy between them and the phenomena of the normal state, and (2) by means of suggestion.

According to Bernheim, hypnotic phenomena are analogous to many normal acts of an automatic, involuntary and unconscious nature; and nothing, absolutely nothing, differentiates natural and artificial sleep. If any distinction at all exists between the normal and the hypnotic state, this

can be explained by means of suggestion. Both the normal and the hypnotised subject can be influenced by it, but, as we have suggested to the latter that he should become more responsive, a peculiar aptitude for transforming the idea into an act has in this way been artificially developed. *In other words, everyone is suggestible, and if you take someone and suggest to him to become more suggestible, that is hypnotism!* Thus, suggestion not only excites the phenomena of hypnosis, but also explains them. Suggestion, *i.e.*, the mental impression, including the preliminary suggestion to become more suggestible, conveyed from the operator to the subject, is the only essential factor in the equation; and the condition of the patient, physical or mental is absolutely unimportant.

This theory contains five distinct propositions, none of which can be accepted without discussion:—

(I.) Nothing differentiates natural and artificial sleep.

(II.) Hypnotic phenomena are analogous to many normal acts of an automatic, involuntary, and unconscious nature.

(III.) An idea has a tendency to generate its actuality.

(IV.) In hypnosis the tendency to accept suggestions is somewhat increased by the action of suggestion itself. Such increased suggestibility, one of degree not of kind, alone marks any difference between the hypnotic and the normal state.

(V.) The result of suggestion in hypnosis is analogous to the result of suggestion in the normal state.

It is also asserted that in the five preceding propositions is to be found a complete explanation of hypnosis and its phenomena.

(1 and 2) *Explanation of Hypnosis by Means of a Supposed General Analogy between it and the Normal Sleeping and Waking States.*

My chief objections to this are:—

(a) That an analogy, no matter how successfully established between two sets of phenomena, by no means explains either of them.

(b) That many important points of difference exist between hypnosis and ordinary sleep.

(c) That the automatic, involuntary and unconscious acts, in which Bernheim seeks to find his analogy, rarely, if ever, occur in hypnosis, and are certainly by no means characteristic of it.

(1) *The Supposed Identity of Hypnosis and Ordinary Sleep.*

Braid considered that marked differences existed between hypnosis on the one hand, and natural somnambulism and the normal sleeping state on the other. These, he thought, consisted mainly in the increased mental and physical powers of the hypnotised subject. Many other authorities agree with Braid. Thus, Moll says, the memory is not at all affected in slight hypnosis; we always presuppose, however, a great decrease of self-consciousness in sleep, and it is just this self-consciousness which remains intact in slight hypnosis.

According to Max Hirsch and Spitta—whose view is shared by Lehmann, the distinguished Danish psychologist—there is no identity between hypnosis and sleep. In reply to Liébeault's assertion that the latter condition results from the concentration of the subject's attention upon the idea of sleep, Hirsch points out that little children fall asleep easily, simply because they do not concentrate their attention, and they, at the same time, are quite unacquainted with the idea of sleep.

Sully finds the following differences between sleep and hypnosis:—(a) The greater part of our dream material in nightly sleep comes from within the organism, and not from without, as in hypnosis. (b) The natural dream is more complex and varied than the hypnotic. (c) The hypnotised subject tries to translate his hallucinations into actions in a manner which finds no parallel in ordinary sleep.

Dèlbœuf says: "I put the subjects to sleep, or more correctly speaking, they believe that they have been asleep." This conception of the condition is, I think, the true one. In deep hypnosis the subjects believe that they have been

asleep, because on awaking they are unable to recall what has happened. The condition, however, may have been characterised by great mental and physical activity, presenting in subsequent amnesia its solitary point of resemblance to normal sleep. Finally, this amnesia itself is not a necessary concomitant of the state, and can be easily prevented by suggestion.

(2) *The supposed Analogy between Hypnotic Phenomena and the Automatic, Involuntary, and Unconscious Acts of Normal Life.*

According to Braid's conception of hypnosis, the state was characterised by mental and physical phenomena, which were not to be found in other conditions. The hypnotised subject had acquired new and varied powers, but had not at the same time lost his volition or moral sense. He asserted that he had proved that no one could be affected by hypnosis, at any stage of the process, unless by voluntary compliance. The subjects were docile and obliging; but, despite this, they refused all criminal suggestions, and even developed a higher sense of propriety than characterised their normal condition.

Totally different views are held by Bernheim and certain other members of the Nancy School. They believe that the subject's volition is weakened or destroyed, and consider this condition to be one of automatism. To both these points I wish to draw your attention.

The hypnotic state is described as one of cerebral automatism; and, according to Bernheim, its phenomena find their analogy in various automatic movements of normal life, such as walking. Suggested crimes, which, I think, differ essentially from acts like these, are described as illustrating automatism in its highest form. It is with the latter that I desire to deal at present. Let us take a typical case. It is suggested to a high-principled girl in the somnambulic state that she shall take a piece of sugar from the basin, and put it into her mother's teacup, after having been informed that this is really a lump of arsenic,

certain to cause death. Apart from the question whether she believes she has committed a real crime, or knows that she has carried out an experiment, let us attempt to understand the supposed mental condition with regard to consciousness and volition, and then compare it with the usual scientific conception of automatism. It is, I think, generally conceded by the Nancy School that hypnotic acts are conscious ones; and that, if amnesia follows on waking, the lost memory can be restored in subsequent hypnoses. Indeed Professor Bernheim, in discussing the Salpêtrière experiments, insists upon the fact that the hypnotised subject is conscious in all stages. The alleged criminal act was, then, a conscious act. Again, we are told that somnambules, before accepting criminal suggestions, frequently struggle against them. This would indicate that, in some instances at all events, they are carried out in opposition to volition as it exists in hypnosis. Further, it must be conceded that a criminal act would not be performed voluntarily by a virtuous person in the normal state. The suggested act, then, is one which would have been opposed by volition in the waking state.

Now let us turn to automatism, as defined by Professor Waller, the well-known physiologist. "The word," he says, "has received two diametrically opposed meanings, viz.: (1) Self-moving, self-rising, spontaneous; (2) automaton-like, that is to say, like a mechanism, that appears to be self-moving, but that we know to be moved by secret springs and hidden keys." The second sense is the one in which he employs it, and is also, I think, the one now generally adopted by science. As the supposed essential characteristic of the suggested crime is the fact that it arose, not spontaneously, but in response to the desires of the operator, it is obvious that if it be automatic at all, it must accord with the second conception of automatism. Now let us follow Dr. Waller in his further definition of the second condition, and then see how this agrees with the so-called hypnotic automatism. According to him, the automatic action is essentially a reflex action, and differs from it only in that it is, as a rule, the habitual or serial effect of

habitual or serial stimuli. An automatic act is the repeated or rhythmic motor response to a repeated or continuous excitation. Usually it is carried on unconsciously. Automatic actions may be divided into two sub-groups: (1) Primary or inherited, of which the act of sucking is an example; (2) secondary or acquired, of which walking is an example. In this discussion I think we may disregard primary or inherited automatic acts. Obviously, to kill one's mother cannot be regarded as an inherited automatism; and, if such a crime be automatic at all, it must fall under the group of secondary ones. Of this form of automatic act the winding of one's watch may be taken as a typical example. This is first performed consciously and voluntarily. After a time consciousness sometimes ceases to participate in the action. On attempting to wind your watch many of you must have occasionally found that you had already done so, although quite incapable of recalling it. Now, this automatic act is simply a voluntary one, performed inattentively or unconsciously; one that has previously been frequently performed, and possesses well-worn nerve channels. It has commenced as a conscious, voluntary act. It has become unconscious by repetition, but still remains voluntary, in the sense that it is an act which the consciousness would approve of did it happen to participate in it.

Let us consider the so-called automatic crime of the hypnotised subject and see how it agrees with this conception of automatism. (1) The crime has not been a habitual one. In the present instance the very nature of the act renders this impossible. Obviously, the subject could not have habitually killed her mother. The alleged automatic act must, then, in this instance, have been performed for the first time. Now the essential character of the secondary or acquired automatic act is that it has been previously, frequently, consciously and voluntarily performed, and possesses in consequence well-worn nerve tracts which offer no obstacle to its fulfilment. (2) The hypnotic act is performed consciously, and the attention, instead of being directed in other channels, as in genuine automatism, is

supposed to be intensely concentrated on the operator, or on the signal given by him—a signal sometimes so faint that the subject is only enabled to recognise it by means of the hyperæsthesia of his special senses. (3) The hypnotic crime is sometimes supposed to be performed in opposition to volition as it exists in hypnosis, and is always supposed to be in opposition to the normal volition. The genuine automatic act, on the other hand, has only been enabled to become automatic owing to the fact that it has previously been frequently performed as a voluntary act; and now, when performed unconsciously, it still remains such an act as the volition, as a general rule, would approve of. The so-called automatic crime of the hypnotised subject not only *differs* then from the general scientific conception of automatism, but is its exact *opposite* in every detail.

While holding, and holding strongly, that the hypnotic crimes, which we are discussing, cannot in any sense be termed automatic, I do not deny that the hypnotic subject can be trained to perform automatic acts. If I suggest some simple movement to a subject, which his volition does not disapprove of, doubtless after a time it may be performed automatically, *i.e.*, after having been frequently, voluntarily and consciously performed, it may at length be executed unconsciously as a genuine automatic act in response to the habitual stimulus which has excited it.

Putting aside all question of the correct conception of the word automatism, or, perhaps better still, characterising the described condition as one of obedience, I now desire to see how far the so-called phenomenon can be taken as the essential characteristic of the hypnotic condition. The writings of the Nancy School indicate a belief that the hypnotic state is essentially characterised by the obedience of the subject to the operator. Some years ago most stress was laid upon complete obedience, and Liébeault said: "We may postulate, as a first principle, that a subject during the state of magnetic sleep, is at the mercy of the hypnotiser." Now, possibly as the result of the influence of the late Professor Delbœuf, a greater power of resistance is conceded. In a recent article, Dr. Liébeault admits that he

has only encountered four to five per cent. of hypnotised subjects to whom one could with absolute certainty suggest crime. This admission is an important one, but despite it, the so-called automatism is still regarded as the essential characteristic of the hypnotic state. If only four or five out of a hundred subjects evince the so-called automatism, surely one is not justified in describing this as the essential characteristic of the whole group. If five out of a hundred un-hypnotised individuals presented certain phenomena which were absent in the remainder, one would surely not choose these rarely occurring phenomena as the descriptive characteristics of the class, and those upon which the belief in other, and more frequently observed phenomena, should depend. Yet, strange as it may seem, this is the position assumed by Durand de Gros, who assured Delbœuf that if he succeeded in proving that the suggested crimes of the Nancy School were recognised by the subject as experimental ones, he would destroy hypnotism entirely. The existence, then, of many undisputed phenomena, which are common to all hypnotic subjects, is to depend upon the acceptance of others which it is alleged occur in four or five per cent. alone out of the same number.

Putting aside in its turn the question of the average number of subjects in whom it is alleged crime can be successfully suggested, I desire first to refer to some of my own observations, and afterwards to consider them in conjunction with the so-called suggested crimes of Bernheim and others, in order to gain, or at all events to strive after, a clearer conception of the mental states involved.

When I commenced hypnotic work some seven years ago, I, like Delbœuf, believed that the hypnotic subject was entirely at the mercy of the operator. I was soon aroused from this dream, however, not by the result of experiments made to test the condition, but from the constantly recurring facts which spontaneously arose in opposition to my preconceived theories. Of these facts the following cases are illustrations :—

Miss C., aged 19, an uneducated girl, had been frequently hypnotised, and was a good somnambule. She suffered greatly

from her teeth, of which she had only sixteen left, all decayed. These were extracted at Leeds in the presence of some sixty medical men and dental surgeons. Anæsthesia was produced by written suggestion, while I remained in another room. The operation was perfectly successful, and unattended by the slightest pain, either then, or afterwards. At a later date I examined her mouth and found that a fragment of one of the stumps remained. I told her to come to my house to have it removed. She mentioned this to one of her neighbours, an old woman, who advised her to have no more teeth extracted, as this would cause her mouth to fall in. The following day she presented herself, and was at once hypnotised, but refused to open her mouth or to permit me to extract the tooth. Emphatic suggestion continued for half an hour produced no result. This was the first occasion on which she had rejected a suggestion. I then awoke her and asked why she refused to have the tooth extracted. She told me what her neighbour had said, and expressed her determination to have nothing more done. I explained the absurdity of this, and pointed out that, as she had only the fragment of one tooth remaining, its removal could not affect the appearance of her face. As she was still obstinate I said: "Unless this fragment is removed you cannot have your artificial teeth fitted." This argument was sufficient. She gave her consent in the waking state, was at once hypnotised and operated on without pain.

Miss D., aged 20, was a good somnambule, who had been the subject of two painless operations. At a later date I wished to satisfy myself of the depth of the hypnotic anæsthesia, as another and more serious operation was contemplated. I obtained her consent to test the condition, hypnotised her and pricked the pulp of the thumb deeply with a needle, and also pinched her arm severely. She showed no sign of pain, and afterwards remembered nothing of what had occurred. A few days later, I wished to repeat the process. She again permitted me to prick her thumb, but when I attempted to pinch her arm she drew it away, and refused to let me touch it. Her mother, who was present, gave the following explanation. After the first experiment, her daughter noticed that her arm was blackened in several places, and asked the cause. When told what I had done, the girl said: "I don't object to being pricked with a needle, but I won't allow Dr. Bramwell to pinch my arm again, because the neighbours might notice the marks." On both occasions her arm was covered, and I did not know it had been marked. I awoke the patient.

She had no recollection of what she had said or done. I told her she had refused to let me pinch her arm, and asked the reason. She laughed, and gave the same explanation as her mother. One day, when I had hypnotised the patient, her mother said to me: "Ask her what she did on a certain occasion." I questioned her, but could obtain no response. I afterwards learnt what she had done. It was something which her mother regarded as a joke, but which was slightly indelicate. Many persons, even fairly refined ones, would have told this without blushing; and I have little doubt the patient would have done so when awake.

Miss E. had been frequently hypnotised and was a good somnambule in whom anæsthesia could be easily induced. She was maid to one of my patients, a chronic invalid, whose house was managed by a sister of uncertain temper. On one occasion, when I had hypnotised E., her mistress requested that I would ask her what had been said to her by this sister. A quarrel had taken place, of a somewhat amusing nature, and my patient wished to hear E.'s account of it in hypnosis; but, despite energetic suggestions, she absolutely refused to say a word on the subject.

Miss F., aged 19, in good health, intelligent, and well-educated. This subject was a very good somnambule in whom anæsthesia and other phenomena of deepest hypnosis could be easily induced. She had a bad memory for words, and was extremely shy in reading, singing, or playing before others. I suggested to her that she should, on awaking, recite some verses with which she was previously unacquainted, and which I had read twice to her when asleep. Shortly after awaking, she repeated them with very few mistakes, and without apparent embarrassment. Her mother assured me that, under ordinary conditions, this feat of memory would have been entirely beyond her power; and that nothing would have induced her to read or recite before me. On another occasion, her mother asked me to suggest during hypnosis, that on waking, she should go to the sideboard in my room, pour out a glass of water, and drink it. This suggestion was not carried out; and was the first which had not been fulfilled. I had taught her mother to hypnotise her; and on returning home, she did so, and questioned her about the suggestion. She recalled it at once, and gave the following reason for not carrying it out, namely, that she did not know me well enough to help herself to a glass of water in my house without being asked.

Mr. G., aged 25, a shopkeeper. He had been frequently hypnotised for medical and surgical purposes, and was a good somnambule. On one occasion I showed him at the York Medical Society. At the close of my lecture I was asked to give an example of changed personality, by making this patient believe he was a dissenting minister preaching a sermon. He refused to do this, and I was then asked to make him believe he was a hawker selling fish. This was also rejected, but he accepted the suggestion that he was Barnum, and that the medical men were wild beasts, and proceeded to describe them in a highly amusing manner. I afterwards tried to make him accept the first two suggestions, but he invariably refused. On one occasion, however, he accepted the suggestion that he should poison a personal friend. The subject was in the alert stage of hypnosis with his eyes open. I took a lump of sugar from the basin and assured him that it was a piece of arsenic sufficient to kill a dozen persons. I then put it in a cup of tea, and told him to give it to his friend to drink. He did so at once. I asked him why he had poisoned his friend, and he replied laughingly, but in an unnaturally gruff voice: "Oh, he has lived long enough." Another young man who was present, also a good somnambule, would carry out suggestions like those rejected by G., but refused to execute the fictitious murder.

The last two subjects, F. and G., accepted suggestions which were apparently in opposition to their normal character. I made no attempt to ascertain G.'s mental condition in reference to the supposed crime, but I think one can without much difficulty imagine it. G. was a respectable tradesman, and a somewhat devout Dissenter; and it is not unnatural to suppose that he refused the part of fish-hawker as this was not in keeping with his social position, and that of minister as it offended his religious susceptibilities, but accepted that of showman because it contained nothing objectionable to him. Would it be reasonable to suppose that he should at the same time be capable of weighing fine distinctions between suggested alterations in personality, and be unable to understand the experimental nature of the crime? He, by the way, affords the only instance in which an imaginary crime has been carried out by one of my own patients. All others, without exception, have absolutely refused such suggestions.

Why should F. have recited the poem and refused to take a glass of water from my sideboard? The answer to the first question is obvious. She was extremely anxious to get rid of the

nervous embarrassment from which she suffered, and thus the suggestion contained nothing opposed to her volition. She herself explained the second.

In the cases above recorded, although a certain amount of evidence was obtained from the patients themselves in reference to their mental condition, no systematic attempt was made to investigate this. I am well aware that this admission is a startling one. I can only say in self-excuse that this all-important point has apparently been equally neglected by others. I have recently attempted to repair the mistake and with interesting results. For example, Miss H., who was the subject of some successful experiments in reference to the appreciation of time in hypnosis, also refused certain suggestions. These I will now relate, as well as her own description of her mental and physical condition during hypnosis. On one occasion during hypnosis, I asked her to put her fingers to her nose at Mr. Barkworth, a friend who assisted at the experiments. She laughed, and despite repeated suggestions, absolutely refused to do so. At a later date, in hypnosis, I asked her for an explanation. She told me she did not want to, and would give no other reason. On another occasion, during hypnosis, I suggested that she should steal Mr. Barkworth's watch. The watch was placed upon the table and Mr. Barkworth hid behind a screen. I told the patient that Mr. Barkworth had gone and had left his watch; that he was very absent-minded, would never remember where he had left it, would never miss it, &c., suggested that she should take it, that no one would ever know, &c. I awoke the patient. She took no notice of the watch. I asked her: "Where is Mr. Barkworth?" "Gone away." "He has left his watch, would you not like to take it?" The patient laughed and said: "No, of course not." I re-hypnotised her and asked: "What did I suggest to you a little while ago when you were asleep?" "That I should steal Mr. Barkworth's watch, that he was absent-minded, would never miss it, &c." "Then why did you not do so?" "Because I did not want to." "Was it because you were afraid of being found out?" "No, not at all, but because I knew it would be wrong."

On another occasion I again questioned her in hypnosis in reference to this suggested theft. I said: "Did you recognise that it was an experiment?" "Yes, perfectly." "How did you know it was?" "I can't tell you, I only felt sure it was." On being questioned further, she said: "Well, I knew you would never really ask me to do anything wrong." "Well, then, if you were quite certain in your own mind that it was only an experi-

ment, why did you not carry it out?" "Because I did not wish to do what was wrong, even in jest." "If, then, I asked you to put a lump of sugar in someone's tea and told you it was arsenic, would you do so?" "No, certainly not." "Not even if you were quite certain that it was only sugar?" "No." "Why?" "Because I should be pretending to commit a crime."

In reply to further questions in hypnosis, she said she felt sure she could refuse any suggestion; that she felt she was herself; that she knew where she was and what she was doing. "Are you the same person asleep as awake?" I asked. "Yes," she replied, with a laugh. She described the condition as a sort of losing herself and yet not losing herself. She knew and heard all that was going on, and yet seemed to take no notice of it. When Mr. Barkworth was put *en rapport* with her, she remembered his voice, and recalled the fact that she had heard it on a previous occasion, when not *en rapport* with him. She said she was resting all the time, and that nothing she did or thought tired her. I asked her what it felt like to have her arm made cataleptic by suggestion? She replied: "I did not feel frightened, but I felt startled. I think it would surprise anyone." "When you wake and find your arm still rigid, what do you feel then?" "I feel amused." "When you are sleeping here, and no one is talking to you, do you ever think of anything?" "Yes! One day I was troubled about my dressmaking. My employer was ill, and I had more responsibility than usual. I had a difficult piece of work to do, and could not understand how it was to be done. When asleep here I planned how I would do it, and carried this out successfully when I returned home. When I awoke I did not know that I had done this. The way out of the difficulty suddenly came into my head on my way home. I now remember planning while asleep what I afterwards carried out."

On one occasion after being hypnotised, and when she was apparently in the lethargic condition, she suddenly volunteered the statement that her mother wished to speak to me. Shortly afterwards Mrs. H. came into the room. The patient was still asleep, and no suggestions of *rapport* were made. Mrs. H. commenced to tell me about a friend in whom she was interested, with a view to finding out whether I thought hypnotic treatment would be of benefit in his case. The patient suddenly joined in the conversation, and added some important details which Mrs. H. had forgotten. On awaking she remembered nothing in reference to this.

On another occasion, under similar circumstances, Mrs. H.

questioned me in reference to a trivial indisposition from which the patient was suffering; and asked me whether I thought she might give her a certain simple remedy. Upon this the patient commenced to laugh, and recounted in a highly amused manner an experiment of her mother's in domestic medicine, of which she had been the unfortunate victim.

At a later date, when questioned regarding the suggested crimes, she gave a somewhat different answer. She said: "I would not take a watch, even if I knew the suggestion was made as an experiment; because this would be pretending to commit a crime. I would, however, put a piece of sugar into a friend's teacup, if I were sure it was sugar, even though someone said it was arsenic; because then I should not be the one who was pretending to commit the crime." So subtle a distinction would not, I think, have occurred to the subject in the waking condition.

I obtained the following account of the hypnotic condition from another patient, an educated, intelligent woman. She said: "When asleep I still feel that I am myself, and can think and reason just as well as when I am awake. I could resist any suggestion if I wanted to do so. The sensation is a pleasant one, as if I were getting rested all over. I am not conscious of other sounds, except your voice. When you are not talking to me, the condition is generally a blank. At such times I occasionally, but rarely, think, or spontaneously recall the events of past hypnoses."

Another educated patient, a very good somnambule, described her state in similar terms. She said: "I feel a kind of restfulness which I do not get in any other condition of life. I feel no fatigue. External sounds, other than your voice, I hear vaguely, as if in a dream, and pay no attention to them. I still feel that I am myself; and can reason just as well as if I were awake." When her arm is made stiff this appears quite natural, as she recognises it is done as an experiment. She feels certain that she could refuse any suggestion which she disapproved of, and would not carry out an imaginary crime, even if she knew it was only an experiment. This patient readily accepted suggestions of anæsthesia and analgesia; and was unable to remember in the waking state either painful sensations or tactile impressions. On being re-hypnotised, however, though she could not recall any sensation of pain, she was able, in response to suggestion, to state where she had been pinched or pricked, and to describe the tactile impressions associated with these operations.

In most of the cases referred to the patients refused to carry out suggestions in hypnosis which they would have rejected in the waking state. Sometimes, however, they refused, in hypnosis, things they would readily have done or submitted to when awake. For example, Dr. Allden, when resident-physician at the Brompton Hospital, hypnotised a girl suffering from chronic pulmonary disease. She rapidly became a good somnambule. On one occasion, after he had hypnotised her, the nurse reminded him that it was his day for examining the patient's chest; but, to his astonishment, she, although naturally docile and obliging, refused to allow it to be bared. She had previously been examined dozens of times by himself and others, and had never made the slightest objection. He insisted upon her submitting, but was unable to overcome her resistance. He asked her why she objected now, when she had never done so previously. She replied: "You never tried before to examine my chest when I was asleep." On awaking she remembered nothing of what had occurred, and he said nothing to her about it. Afterwards the nurse told her, whereupon she was greatly distressed; and wondered how she could possibly have been so rude to the doctor.

These, and many similar facts, have forced me to abandon all belief in the so-called automatism, or helpless obedience; still I must refer to some of the arguments in support of it, before attempting to analyse further the mental condition in hypnosis. Of these arguments the following are examples:—

(a) When subjects successfully resist suggestion, it is usual to explain this by assuming that they have not been so deeply hypnotised as those in whom no resistance has manifested itself. I cannot admit the correctness of this in my cases. During the last seven years, I have had frequent opportunities of examining hypnotic subjects, at home and abroad; and have nowhere observed more profound somnambules than amongst my own patients—rarely in fact seeing cases to equal them. All to whom I have referred not only exhibited the phenomena of profoundest somnambulism, but nearly all had been subjects of painless operations in the hypnotic state.

(b) The personality of the operator, and his method of training his subjects, is supposed to play an important part in the acceptance or rejection of suggestions. Granting that this be true, it does not explain the resistance which I encountered. I commenced by believing that the subjects were entirely at my mercy, and did my best to develop their supposed obedience.

(c) The existence of one class of phenomena is considered as necessarily implying the existence of another and totally differing class. Durand de Gros asks: "Is it possible that suggestion should have the power of producing extraordinary physical changes, and yet be without this particular effect upon the moral state?" The facts I have already cited answer this question in the affirmative. Putting these aside, the assumption that the physical phenomena necessarily imply certain moral ones is unreasonable. What inevitable connection exists, for example, between an alteration in the pulse rate and the murder of one's mother? Should I not be equally logically justified in assuming that the subject in the normal state who, in response to suggestion, would play the violin or paint a picture, would be equally willing to rob a church?

(d) Evidence in favour of obedience afforded by cases in which the subjects are alleged to have accepted criminal and analogous suggestions. These are important. The fact that the phenomenon of helpless obedience was invariably absent in my patients does not justify me in concluding that it did not sometimes occur in those of others. These cases of so-called automatism fall into two classes: (1) Where an imaginary crime has been suggested; (2) where a real act has been performed, which it is assumed the patient would not have submitted to in the normal state.

(1) First, as regards imaginary crime; here, as Professor Delbœuf has pointed out, it is supposed that the patient is passing through a mental state similar to that of the operator. Assumption without experimental proof is a frail and unsatisfactory basis on which to erect a theory. Let us first examine the facts. A somnambule puts a piece of sugar

into her mother's teacup, while her medical man makes various absurd and untruthful assertions as to its composition. Bernheim and Liégeois believe that the subject accepted these absurd statements as true because, being hypnotised, she was unable to distinguish between truth and falsehood, while Delbœuf claims that she had sufficient sense left to know exactly what she was doing. *To neither does it seem to have occurred to ask the subject during hypnosis what she thought about the matter herself.* If they had done so, she would have promptly solved the difficulty, and told them that while they were discussing probabilities, she was quietly laughing in her sleeve at the grotesque absurdity of the whole performance. It may be noticed in passing that while Bernheim considers the Salpêtrière subjects so abnormally acute that they can catch the slightest indication of the thoughts of the operator, and so destroy the supposed value of the phenomena alleged to be induced by metals, magnets, drugs in sealed tubes, &c.; he, on the other hand, supposes the Nancy subjects to be so abnormally devoid of all intelligence as to be unable to understand when a palpable farce is played before them.

(2) Where a real act has been performed which it is assumed the patient would not have submitted to in the normal state. Of this Bernheim cites an example. He states that he uncovered a young woman, presumably a hospital patient, in the presence of his assistants, and that she appeared perfectly calm and indifferent. Bernheim also quotes a case in which he entirely failed in persuading another young woman, also a good somnambule, to allow him to uncover her or to commit an imaginary crime, despite the fact that he varied the suggestion in every conceivable way. Let us take the case of supposed helpless obedience. The mere fact that a woman permitted herself to be uncovered does not necessarily imply that she was incapable of resisting. Before this can be used as an argument in favour of the helpless obedience of the hypnotised subject, one is justified in demanding that it should be clearly proved that, under similar circumstances, the patient would have objected to being uncovered in the waking condition. Medical men are frequently obliged to uncover their female

patients for examination, and rarely encounter resistance. The first time I visited Professor Bernheim's wards I was struck by the fact that, in order to show some hysterical muscular movements in the abdomen of a non-hypnotised subject, he threw off the bed clothes, drew the patient's nightgown up to her neck, and left her in that condition while we examined other cases. Though such treatment is opposed to the practice of English hospitals, I do not propose to criticise it. I only desire to draw attention to the fact that a patient was stripped in the waking state, from my point of view unnecessarily so, and that she and everyone else apparently regarded this as devoid of importance. The hypnotised subject did not object to be stripped. Why should she? She must have been accustomed to see other patients examined, and, apparently, there was no special reason why she should have objected to the ordinary routine. Cases such as these appear to me absolutely valueless, while the reasons for the patient's supposed obedience remain so easily accounted for in other ways. To render such a case worthy of serious consideration it would be absolutely necessary to eliminate many important factors such as: (a) The fact that the subject was ill. (b) That she was in a hospital where patients were stripped as part of the ordinary routine. (c) That the examination was made by a medical man.

Strangely enough the most marked case of resistance to suggestion that I have observed was that shown by Liébeault's celebrated somnambule, Camille. When I first visited Nancy in 1889, Dr. Liébeault showed me this subject, who had been frequently hypnotised, and whom he regarded as a typical specimen of profound somnambulism illustrating hypnotic automatism in its highest degree. He assured me that the suggestions he made to her were carried out with the fatality of a falling stone. He hypnotised her and suggested that on awaking she should find on opening the outer door that there was a violent snow-storm; that she should at once return, complain of this, and proceed to the stove to warm herself. While so doing one of her hands would touch the stove and she would believe that she had burnt it. It was a warm summer's day, and, of course, the

stove had not been lighted. The patient refused to accept the suggestion. Dr. Liébeault insisted for some time, and then gave up the attempt, saying that she sometimes refused suggestions. He then asked her: "Will you do this another time if you will not do it to-day?" She replied: "Yes, to-morrow." On the following day the suggestion was repeated and carried out in all its details. In this instance, then, the classic hypnotic automaton, the one who was supposed to carry out a suggestion with the fatality of a falling stone, refused one, not on moral grounds, but apparently from pure caprice.

The difference between the hypnotised and the normal subject, is to be found, not so much in conduct as in the increased mental and physical powers of the former. Any changes in the moral sense that I have noticed, have invariably been in favour of the hypnotised subject. As regards obedience to suggestion, there is apparently little to choose between the two. A hypnotised subject, who has acquired the power of manifesting various physical and mental phenomena, will do so in response to suggestion, for much the same reasons as one in the normal condition. In the normal state we are usually pleased to show off our various gifts and attainments, more especially if we think they are superior to those of others, and in this respect the hypnotised subject does not differ. Both will refuse what is disagreeable; in both this refusal may be modified or overcome by appeals to the reason, or to the usual motives which influence conduct. When the act demanded is contrary to the moral sense, it is usually refused by the normal subject, and invariably by the hypnotised one. I have never observed any decrease of intelligence in hypnosis. In the alert stage it is often conspicuously increased, while in the lethargic it is only apparently, not really, suspended. Forel's warders, who could sleep by the bedside of suicidal maniacs, and awake immediately at a given signal; or, who could inhibit their own hearing of the purposeless noises of the insane, and acutely hear everything which demanded their attention, did not in so doing show any loss of intelligence. The power of concentration in the normal state, with its accompanying

inhibition of undesirable impressions, is a well-known and somewhat analogous condition, but one which is not usually regarded as indicative of mental degeneration.

When one turns to the later works of Braid, and sees how clear was his experimental proof that the hypnotised subject not only had the power of choosing between suggestions, but invariably refused those repugnant to his moral nature, one cannot help feeling surprised at the revival of theories, in reference to so-called automatism or obedience, identical with the views of the mesmerists. More especially so when one considers that Bernheim, who holds these views, also boldly asserts that there is nothing in hypnotism but the name; that it does not create a new condition, and that hypnotic acts are only exaggerated normal ones. According to Bernheim, however, the moral state in hypnosis differs widely from the normal; and this is in obvious contradiction to his own conception of hypnotism. One can understand, for example, how a prolonged muscular rigidity may be a hypnotic exaggeration of a somewhat shorter normal one; but it is difficult to comprehend how the murder of one's mother when hypnotised, can be an exaggeration of the *refusal* to hurt a fly when awake. Bernheim's view of the moral state does not follow logically from the supposed resemblance between the hypnotic and normal condition, but apparently has its origin in an erroneous estimate of the nature and power of suggestion. On the one hand, he tells us that the hypnotic and normal conditions are practically identical; their only distinction a slight difference in suggestibility. On the other hand, we are informed that a virtuous individual will commit crime in response to hypnotic suggestion. If this were correct, we should be justified in describing the origin of hypnotic crime thus:—

(a) A virtuous girl in the normal state has a natural tendency to accept the suggestion that she should murder her mother.

(b) In hypnosis suggestibility is slightly increased and thus, when it is suggested to her to murder her mother, she does so.

The views which I have just expressed, regarding the

hypnotised subject's power of rejecting disagreeable or criminal suggestions, are shared, more or less completely, by a good many other observers. They, however, as far as I have been able to learn, base their statements solely on cases where suggestions have been rejected, without attempting to examine the patient's mental state, by questioning them in hypnosis.

Thus, Professor Beaunis says hypnotised subjects usually reason very logically and evince striking powers of deduction; they are certainly not unconscious machines incapable of judgment. Beaunis frequently observed that subjects refused suggestions which were disagreeable to them.

Dr. Crocq, of Brussels, also admits that subjects frequently reject distasteful suggestions.

Richer believes that a somnambule may entirely refuse to perform certain acts and oppose no resistance to others.

According to Gilles de la Tourette, the hypnotic somnambule is not a pure automaton, a simple machine that one can turn at will. He possesses a personality, sometimes, it is true, subdued or weakened, but which in certain cases persists in its entirety, and shows itself clearly by the resistance it opposes to suggested ideas. The hypnotised subject always retains his individuality and can manifest his will by resisting suggestions.

Brouardel says that if agreeable or indifferent suggestions are made to a somnambule by a person whom he likes and trusts, he will accept them; but, if they are contrary to his personal affections or his natural instincts, he opposes an almost invincible resistance to them.

Pitres mentions a case of a somnambule who refused to awake whenever a disagreeable post-hypnotic suggestion was made to him.

According to Dr. Charpignon it is much easier to restore moral rectitude, by means of hypnotic suggestion, to a somnambule who has lost it, than to pervert, by the same means, a person of high moral character.

Dr. de Jong cites many cases of resistance to suggestion. One of his patients, a profound somnambule, appar-

ently accepted criminal suggestions as readily as she did innocent ones. She refused, however, to undress before another person, and would not tell an insignificant secret that she had promised not to reveal. Another patient refused to tell Dr. de Jong something of an indelicate nature which she had seen, and which she had already confided to a friend of her own sex. These subjects, according to de Jong, refused certain suggestions, because they involved real facts which were disagreeable to them, while they executed others alleged to be criminal, because they recognised that these were laboratory experiments devoid of danger either to themselves or others.

Delbœuf relates an experiment with J., an excellent somnambule. I shall again refer to this patient in speaking of two symmetrical burns. At a later date, during hypnosis, she was painlessly delivered of her first child by Dr. Fraipont of Liège. J. was a courageous young woman and sometimes took care of Delbœuf's country house in his absence. She was armed with a revolver, owing to the serious strikes and riots existing at that time. One night a man tried to force the door. The barking of the dogs awakened J.; she opened the window, saw the man, took her revolver, went into the hall, and watched for his entry in order to fire at him. The man, possibly frightened by the noise, disappeared. On a subsequent occasion, Delbœuf secretly discharged the revolver; then hypnotised J., and suggested that two persons in another room were robbers, and insisted that she should fire at them. In obedience to his suggestions she fetched the revolver, but, despite his reiterated and emphatic commands, absolutely refused to fire; on the contrary, she stepped backwards and placed the revolver cautiously on the floor.

Suggested Crimes.—Summary.

(1) I have never seen a suggestion accepted in hypnosis which would have been refused in the normal state.

(2) I have observed that suggestions could be resisted as easily in the lethargic as in the alert stage.

(3) I have frequently noticed increased refinement in hypnosis; subjects have refused suggestions which they would have accepted in the normal condition.

(4) I saw Camille refuse a suggestion from mere caprice.

(5) Examination of the mental condition in hypnosis revealed the fact that it was unimpaired.

(6) The arguments of Bernheim are devoid of value as they are founded exclusively on cases where (a) a simple and harmless act has been assumed to be thought criminal by the subject, because the operator has stated it to be so, and (b) where the subject has permitted something in hypnosis, which he would probably have submitted to in the normal state.

(3) *An Idea has a Tendency to Generate its Actuality.*

According to Bernheim, the suggestive phenomena of hypnosis depend upon the fact that, in the normal subject, an idea has a tendency to generate its actuality. This power, as we shall see, is supposed, and erroneously so, to be artificially increased by suggestion in the hypnotised subject alone.

Now, if we confined our attention to the hypnotic state and considered how frequently a suggested idea, unassociated with violent emotional conditions, produced a rapid and definite response, we should be inclined to admit that in hypnosis an idea not only had a tendency to generate its actuality, but almost invariably did so.

A similar statement, however, in reference to normal life, cannot be accepted without question. If an extended statistical inquiry were made as to the results of suggestion we should find that these would fall under three classes.

(1) Where the suggested idea had produced no result.

(2) Where the result was opposite or, at all events, different from that intended.

(3) Where the suggestion had been responded to with more or less exactitude.

(I.) *Where the Suggested Idea produced no Result.*

A very casual glance at the events of everyday life would compel us to conclude that this class is the commonest of

the three. This is evident, if we think of the numberless things ineffectually suggested in the family circle, to domestics, workmen, tradespeople, friends, acquaintances, &c.

(II.) *Where the Result produced was Opposite or, at all events, Different from that Intended.*

Numerous examples of this class could easily be cited. Thus, if a thief snatches my watch and runs away, and I suggest to him to stop, he, on the contrary, runs the faster. If a street arab is making a noise under my window and I tell him to cease and go away, he not only persists, but incites others to join him.

(III.) *Where the Suggestion has been Responded to with more or less Exactitude.*

When we compare the results of this class with those obtained in the others they must, I am afraid, sink into insignificance. For one suggestion which has generated its actuality we must count at least a hundred which have produced nothing, and possibly ten where the result has been an unexpected or disagreeable one. I would, therefore, re-state the proposition as follows:—

(a) A suggested idea has generally a tendency to generate nothing.

(b) A suggested idea has frequently a tendency to generate its opposite.

(c) A suggested idea rarely tends to generate its actuality.

When the theory first crept into psychology that a suggested idea had a greater tendency to evoke its actuality than to produce other or negative results, I do not know, but certainly it is now frequently quoted as an accepted truth. The production of a flow of saliva by the sight of food is the stock illustration of the alleged phenomenon. The most important factor in the equation—hunger in the subject of the experiment—is apt to be lost sight of. It is quite possible that the sight of a couple of pounds of raw beefsteak may produce a flow of saliva in a very hungry

man. But, after these had been cooked and eaten, if he were shown a similar piece of raw steak, its sight, instead of inducing a flow of saliva, would in all probability simply evoke feelings of disgust.

To this point, namely, that the result of an idea does not depend so much upon the idea itself as upon the nature of its recipient, I will again refer when discussing "Suggestion." Meanwhile, I wish to emphasize the fact that "tendency" implies numerical proportion. For example, if I fire a dozen times at a target, and if on five occasions the bullet strikes it, and on seven misses and passes to the right, the *tendency* must be in favour of the results which are numerically the greater of the two. In the same way, before concluding that an idea has a tendency to generate its actuality, one must be able to prove that out of a given number of cases this result follows more frequently than others.

(IV.) *In Hypnosis the Tendency to accept Suggestions is somewhat increased by the Action of Suggestion itself; this alone distinguishes the Hypnotic from the Normal State.*

Granting for the moment that the normal and hypnotic states are practically identical; that both are characterised by susceptibility to suggestion; how far are we justified in concluding that increased suggestibility is possessed by the hypnotic subject alone, and affords the only difference which distinguishes him from the normal one? Let us consider the means by which the change has been stated to have been brought about. The phrase: "You are to become more suggestible," is supposed to have artificially created it. Now, if we admit that this formula is sufficient to change a normal into a hypnotic subject, and to account for his increased suggestibility, we must be prepared to show, in order to maintain the distinction between the two, that the individual who is still regarded as normal *i.e.*, less suggestible, has escaped similar influences. Suggestions in normal life, however, are frequently associated with those of increased suggestibility. A beggar, in appealing for alms,

not only asks that they should be given him, but also suggests in various ways, directly or indirectly, according to his skill and ingenuity, that the object of his petition should become more responsive to his prayer, *i.e.*, more suggestible. There is an important difference between the two. In hypnosis we attempt to gain increased power by quietly repeating some recognised formula once or twice, while in normal life we attempt to obtain it in a much more forcible and varied manner. We must conclude, then, that if the hypnotic and normal states are practically identical, and suggestion a factor common to both, suggestibility, as the result of the methods employed to develop it, ought to be more markedly characteristic of the normal than of the hypnotic condition.

(V.) *The Result of Suggestion in Hypnosis is Analogous to the Result of Suggestion in the Normal State.*

If we confine ourselves to cases in which suggestion in the normal subject has been employed in the same manner as it is used in hypnosis, the analogy is at once seen to be an extremely imperfect one. The results of the workings of the mind acting, so to say, in cold blood upon the body, are extremely rare and generally unimportant. On the other hand, if we turn to the effects of violent emotional states, we find many phenomena have been produced by them which more or less closely resemble the phenomena of hypnosis. Similarity of result, however, does not necessarily imply identity of cause; and an attempted analogy which is based solely on the former and ignores the latter must ever be an imperfect one. Fear, hope, faith, or religious excitement is almost invariably present in cases which are cited as analogous to hypnotic ones. Not only are these conditions unnecessary for the induction of hypnosis, but some of them absolutely preclude its production. Thus, hypnotic phenomena can be evoked in the absence of all those conditions that are essential for the production of similar phenomena in the normal state; and, further, the presence of some of these conditions, instead of favouring hypnosis, hinders or

prevents it. Putting aside this important objection—the difference between the conditions associated with the development of the phenomena—there still remain certain points of contrast between the phenomena themselves, which I shall now enumerate.

(A) *Suggestion in Hypnosis.*

(I.) Once hypnosis has been induced, a wide range of phenomena, both mental and physical, can be evoked at any time, and with the consent of the subject by anyone. A considerable number of phenomena can be simultaneously produced in the same subject.

(II.) One phenomenon can be immediately changed into its opposite, *i.e.*, muscular rigidity into paralysis, anæsthesia into hyperæsthesia, &c.

(III.) Hypnotic phenomena can be terminated at will.

(IV.) The date of the appearance of the phenomenon can be delayed, *i.e.*, it can be suggested during hypnosis that it shall not appear till twelve months afterwards.

(V.) The suggestion will invariably be responded to subject to two important limitations, *i.e.*, that it contains nothing in opposition to the patient's moral sense, and that it is not beyond the range of his hypnotic powers.

(VI.) Under the conditions just mentioned, the exact nature of the response can be predicted, *i.e.*, similar stimuli produce identical results.

(VII.) Patients who readily respond to suggestions when hypnotised are frequently the very subjects who have for years resisted suggestion in the normal condition, even when this has been associated with emotional states. For example, a patient who had long suffered from dipsomania received many and varied suggestions in the normal state. The grief of his friends and relatives and their repeated remonstrances were powerful suggestions. So, too, were the loss of fortune and self-respect, and the physical sufferings, associated with keen remorse, which followed his drinking bouts. Twelve months passed in a home for inebriates must also have been full of suggestions of many kinds. All these, however, produced no result; and yet, after a few weeks' hypnotic treat-

ment, the patient abandoned the alcoholic habit, and still, after the lapse of six years, remains an abstainer.

(VIII.) Hypnotic suggestion tends to gain strength by repetition.

(B) Suggestion by Means of Emotional States.

(I.) The resultant phenomena are usually isolated ones, or, at all events, much more limited in number than those which can be simultaneously evoked in the hypnotised subject. Further, they cannot be produced by anyone. Thus, the subject who had been influenced by the "touch" of a king, would probably be unresponsive to that of a peasant.

(II.) One phenomenon cannot be immediately changed into its opposite without an alteration in the emotional state which had produced it.

(III.) Emotional phenomena cannot be terminated at will.

(IV.) The date of the appearance of the phenomena can rarely be delayed and fixed.

(V.) The phenomena are evoked with less certainty than in hypnosis; an emotional state which will produce a physical effect in one subject will produce nothing in another.

(VI.) Identical emotional states do not always produce similar physical phenomena. On the contrary, opposite conditions are frequently evoked in different subjects by identical emotions; *e.g.*, fear will paralyse one, and excite violent muscular movements in another.

(VII.) Patients who refuse suggestions in the normal state readily accept similar ones when hypnotised.

(VIII.) An emotional suggestion frequently loses strength by repetition, *i.e.*, a subject may readily come to disregard former fears.

So far we have been occupied in discussing the facts upon which Bernheim's theoretical explanation of hypnosis is founded. To these exception has been taken on every point, *viz.* :—

(1) To the supposed identity of normal and hypnotic sleep.

(2) To the supposed analogy between the phenomena of hypnosis and automatic involuntary and unconscious acts of normal life.

(3) To the general principle that, in normal life, an idea has a tendency to generate its actuality.

(4) To the statement that suggestion increases suggestibility in the hypnotised subject alone.

(5) To the supposed general analogy between the result of suggestion in the normal state and in hypnosis.

In all these statements there is only one I am prepared to accept, namely, that suggestion plays an important part in evoking hypnotic phenomena. There remains for our consideration the question how far suggestion *explains* the phenomena of hypnosis.

Does Suggestion explain Hypnosis and its Phenomena?

The answer to this question must, I think, be a distinctly negative one. The success of suggestion depends, not on the suggestion itself, but on conditions inherent in the subject. These are: (1) Willingness to accept and carry out the suggestion, and (2) the power to do so. In the hypnotised subject, except in reference to criminal or improper suggestions, the first condition is generally present. The second varies according to the depth of the hypnosis and the personality of the patient. For instance, I might suggest analgesia, in precisely similar terms, to three subjects, and yet obtain quite different results. One might become profoundly analgesic, the second slightly so, and the third not at all. Just in the same way, if three jockeys attempt to make their horses gallop a certain distance in a given time, the suggestions conveyed by voice, spur, and whip may be similar, and yet the results quite different. One horse, in response to suggestion, may easily cover the required distance in the allotted time: it was both able and willing to perform the feat. The second, in response to somewhat increased suggestion, may nearly do so: it was willing, but had not sufficient staying power. The third, able but unwilling, not only refuses to begin the

race, but bolts off in the contrary direction. With this horse we have the exact opposite of the result obtained in the first instance; and yet possibly the amount of suggestion it received largely exceeded that administered to the others. As Myers has pointed out, the operator directs the condition upon which hypnotic phenomena depend, but does not create it. "Professor Bernheim's command, 'Feel pain no more,' is no more a scientific instruction *how* not to feel pain, than the prophet's 'Wash in Jordan and be clean' was a pharmacopœial prescription for leprosy." In hypnosis, the essential condition is not the means used to excite the phenomena, but the peculiar state which enables them to be evoked. Suggestion no more explains the phenomena of hypnotism than the crack of a pistol explains a boat race. Both are simply signals—mere points of departure, and nothing more. In Bernheim's hands the word suggestion has acquired an entirely new signification, and differs only in name from the "odyllic" force of the mesmerists. It has become mysterious and all-powerful, and is supposed to be capable, not only of evoking and explaining all the phenomena of hypnotism, but also of originating—nay, even of being—the condition itself. According to this view, suggestion not only starts the race, but also creates the rowers and builds the boat!

Braid's Views regarding Suggestion.

While Braid held that hypnotic phenomena resulted from dominant ideas in the mind of the subject, he, at the same time, stated that it was a matter of indifference whether these had existed previously, or were afterwards audibly suggested by the operator, or indirectly created by the sensory impressions resulting from his manipulations.

(I). Self-suggestion.

Braid cites many instances in which hypnosis and its phenomena were entirely the result of self-suggestion, although they were supposed to be due to other causes.

(II.) *Passes and other Manipulations.*

According to Braid, everything which produces a new impression is capable of modifying existing functions, whether the impression be of a mental or a physical nature. Hence he thought it possible that passes might have a physical effect through mechanical agitation of the air, by touch, or by producing some change in temperature or in electrical states. In support of this he drew attention to the fact that physical impressions, too slight to arouse consciousness, might yet produce potent results; for example, although a person did not notice he was sitting in a draught, this might cause rheumatism. He considered that physical methods acted most powerfully, however, when they directly excited mental action, either by fixing the attention on one part or function of the body, and withdrawing it from others, or by arousing ideas previously associated with the physical impression. In reference to the latter point, he says, this is simply an inversion of the usual sequence existing between mental and muscular excitation. Under ordinary circumstances the mental impression precedes and excites the physical one, but here the order of events is reversed. The touch calls into play the muscles constituting the "anatomy of expression," of any given passion or emotion, idea or train of thought; and, secondly, this physical expression suggests to the mind of the subject the corresponding idea with which it has been previously associated in the waking condition. Though Braid explained the action of passes in this way, he also drew attention to the fact that their effects might be neutralised, or even reversed, by direct suggestion. Thus, he said, suppose the operator, while making the passes, spoke aloud and predicted what would happen, the *verbal suggestion* might be so strong as to cause the predicted manifestation to be realised instead of what otherwise would have been the case; and thus, from this time forward, through the hypnotic memory, a similar impression on that part or organ of sense would recall the previously

associated idea and manifestation. Though Braid at first employed passes, &c., he almost entirely discarded these methods when he began to regard hypnotic phenomena from a psychical standpoint. He then stated distinctly that the value of mechanical means was purely suggestive, and that direct verbal suggestion was best for exciting the phenomena of hypnosis, whether experimental or therapeutic. His usual method was as follows: After hypnotising his patients, he stated, in a confident manner, the results he wished to obtain, and, in certain subjects, found that these could be varied by simple change in the voice. Thus, if he made a patient see an imaginary sheep, and then asked him in a cheerful manner what colour it was, this tone usually elicited the reply, "White," or some light colour. If he then asked: "What colour is it now?" giving a sad intonation to the word *now*, the reply would usually be, "Black."

(III.) *Points of Difference between Braid and the Nancy School as to Suggestion.*

The difference between Braid and the Nancy School, with regard to suggestion, is entirely one of theory, not of practice. Braid employed verbal suggestion in hypnosis just as constantly and just as intelligently as any member of the Nancy School. This fact is denied by Bernheim, who says: "It is strange that Braid did not think of applying suggestion in its most natural form—suggestion by speech—to bring about hypnosis and its therapeutic effects. He did not dream of explaining the curative effects of hypnotism by means of the psychical influence of suggestion, but made use of suggestion without knowing it." This statement has its sole origin in ignorance of Braid's later works. In these his references to the use of verbal suggestion in therapeutics are both clear and numerous. For example, in describing his method of treating cases of hysterical paralysis, Braid insists upon the importance of removing morbid ideas and substituting salutary ones of vigour and self-confidence in their place. This, he says,

is to be done "by *audible suggestions* addressed to the patient, in a confident tone of voice, as to what must and shall be realised by the processes he has been subjected to." Again, he says: "By various modes of *suggestion*, either by *audible language* spoken within the hearing of the patient, or by definite physical impressions, certain ideas are fixed strongly and involuntarily in his mind."

Braid, however, while agreeing with Bernheim as to the practical use of suggestion, differs entirely from him in his theoretical conception of it. He did not consider suggestion as explanatory of hypnotic phenomena, but, like Myers at a later date, regarded it simply as an artifice used in order to excite them. He considered that the mental phenomena were only rendered possible by previous physical changes and, as the result of these, the operator was enabled to act like an engineer, and to direct the forces which existed in the subject's own body.

(IV.) *The Views of Braid and of Forel as regards Passes.*

While Braid believed that the mental effect resulting from the indirect physical action of mechanical means could be checked or reversed by stronger and more direct verbal suggestion, he still held, and I think justly so, that physical impressions were capable of producing both physical and mental results. Forel, on the other hand, denies the physical influence of mechanical processes, on the ground that suggestion is capable of altering their supposed action. He says: "Blowing on the face no longer awakens my subjects, because I have suggested that this would remove pain instead of arousing them." From this he concludes that the act of blowing produces no result, and considers this a powerful argument against the Somatic School. Would it not be equally logical to contend that the prick of a pin produced no physical effect because the patient, rendered insensible to pain by suggestion, had been taught to regard the pin-prick as a signal to evoke some other condition?

Attention in Hypnosis.

While Braid and Bernheim differ as to the physical changes which precede or accompany hypnosis, they both attach much importance to the question of attention in reference to the induction of hypnosis and its phenomena.

(I.) Attention in the Induction of Hypnosis.

According to Braid, the induction of hypnosis was facilitated either by :—(1) The concentration of the attention on some external object ; (2) concentration of the attention on some idea connected with hypnotism.

Liébeault and Bernheim consider that ordinary and hypnotic sleep are both due to the fixation of the attention and of the nervous force upon the idea of sleep. The individual, they say, who desires to go to sleep chooses a quiet spot, meditates and keeps still. His nervous force is concentrated upon a single idea, and deserts the nerves of sensation emotion and special sense. The conditions which induce the hypnotic state are identical. The subject is told to concentrate his mind upon the idea of sleep ; and, to aid him in doing so, is directed to look fixedly at some object. From this results bodily repose, the senses become less acute, more and more isolated from the external world, and, finally, thought is arrested.

A connection undoubtedly exists between the subject's power of attention and the facility with which hypnosis can be induced. For example, idiots, who possess little spontaneous and no voluntary attention, cannot be hypnotised at all, and others, such as those suffering from mania, hysteria, &c., whose attention is actively turned into other channels, are extremely difficult to influence.

In reference to the connection between the attention and the induction of hypnosis, the following points seem worthy of notice:—

(a) It is not necessary that the attention should be concentrated on the idea of sleep. Braid, as we have seen, easily induced hypnosis when the patient gazed steadily at

an external object, and concentrated his attention on the idea of that object. The primary hypnosis also need not resemble sleep, and the subject may at once pass into the alert stage with the eyes open.

(b) Primary hypnosis has sometimes been induced in cases where it would be difficult to prove that any concentration of attention had existed, either upon some external object, or upon the idea of sleep. In these cases, the subject, after having given his consent to the experiment, has rested quietly, and voluntarily reduced his mental activity. He has, as nearly as possible, emptied his mind of all thought, and produced, not a condition of concentration, but its opposite—abstraction.

(c) The phenomena of natural somnambulism, which, as Gurney points out, in respect to the absorption of the mind in one direction, present the closest analogy to those of hypnotism, demand no previous concentration of attention at all.

(d) Once hypnosis has been induced, the condition can be evoked at any time, and practically instantaneously, in response to a previously arranged signal. Here, then, although the attention of the subject has been momentarily directed to the signal, prolonged concentration of attention has been absent.

(II.) *Does Concentration of Attention cause Hypnosis?*

Gurney said: "But, even if we confine ourselves to cases where attention is actually present during the production of the state, what ground is there for describing it as the cause of that state? The general effects of a one-sided strain of mind or body are pretty well-known, and 'tonic cramp of the attention' may be a very satisfactory description of the one-sided absorption in a particular direction which characterises many isolated stages of hypnotic trance. But what tendency should the cramp of an attention, which is directed to a button held in the hand, have to produce, or to facilitate, a fresh cramp or series of cramps, when the attention is diverted to quite fresh objects? I have again and again

found the complete change to a new genus of ideas to be absolutely effortless and instantaneous—found, that is, that the attention, which has been, as usual, fixed during the process of hypnotisation, became quite abnormally mobile afterwards. This great mobility of attention seems an odd result of previous rigid attention to a button. If I am told that a particular mental attitude—that of fixed or one-sided attention—is the cause of certain mental phenomena which are new to me, I am surely justified in demanding that the order of events shall present some perceptible coherence—shall at least not run directly counter to what my general experience will have led me to expect.”

Again, taking the case where the attention was concentrated during the production of the state, how does this explain the fact that, when the patient is left to himself in hypnosis, his condition is usually one of abstraction? Here, then, as a supposed result of a previous concentration of attention, we have the spontaneous development of its opposite.

Preliminary fixation of attention cannot be accepted as an explanation of subsequent mobility. All that we can concede so far is :—(1) That fixation of the attention frequently precedes, and usually facilitates, the induction of hypnosis. (2) That the attention in hypnosis can easily be rendered excessively mobile.

(III.) *Rapport and Attention.*

The following is Liébeault's view : “ It is observed,” he says, “ that nearly all artificial somnambules are in relation by their senses with those who put them to sleep, but only with them. The subject hears everything the operator says to him, but him only, provided the sleep is sufficiently deep. He only hears the operator when he is addressed directly by him, and not when a third person is spoken to. This *rapport* extends to the other senses.” According to Liébeault, the subject remains *en rapport* with the hypnotiser because he goes to sleep while thinking of him, and this does not differ from what sometimes happens in ordinary

sleep. A mother who goes to sleep close to the cradle of her child does not cease to watch over him during her sleep, but, while she hears his slightest cry, is insensible to other louder sounds. The concentration of the subject's attention upon the operator, and his mental retention of the idea of the one who put him to sleep, is the cause of *rapport*.

In *rapport*, Bernheim finds his solitary point of difference between normal and hypnotic sleep. He says: "In ordinary sleep, as soon as consciousness is lost, the subject is only in relationship with himself; in induced sleep his mind retains the memory of the person who has put him to sleep, hence the hypnotiser's power of playing upon his imagination, of suggesting dreams, and of directing the acts which are no longer controlled by the weakened or absent will."

The following is Braid's view of the condition of the attention in hypnosis:—

"The principal difference between hypnotic and normal sleep is to be found in the mental condition. When falling into ordinary sleep the mind passes from one idea to another indifferently, and the subject is unable to fix his attention on any regular train of thought, or to perform any act requiring much voluntary effort. As the result of this, audible suggestions and sensory impressions received by the sleeper, if not intense enough to entirely awaken him, seldom do more than arouse dreams, in which ideas pass through his mind without exciting definite physical acts. On the other hand, the concentration of attention, which is the result of the means employed for inducing hypnosis, is continued into the state itself, and verbal suggestions or sensory impressions excite definite trains of thought or physical movements, instead of dreams."

Certain points of difference and resemblance are to be noticed between these views.

According to Braid, the condition of the attention in hypnosis favoured response to external suggestion, but not to suggestion conveyed by any particular person, such as the hypnotiser. It was possible by suggestion to create an artificial state in which the subject was apparently only *en rapport* with the operator, but this condition was only an

apparent, not a real one. The subjects really heard the suggestions of others, though special artifices might be necessary in order to make them respond to them. Braid relates a case in which he made a somnambule respond to his indirect suggestions, conveyed in the form of confident predictions of what was going to happen, though the subject was asleep when he entered the room, and apparently was only *en rapport* with the original operator. Carpenter drew attention to the fact that *rappport* was unknown to Mesmer and his immediate disciples, and was not discovered until long after the practice of mesmerism had come into vogue. The phenomena of *rappport* only acquired constancy and fixity in proportion as its laws were announced and received. Mesmerists ignorant of *rappport* produced a great variety of remarkable phenomena, and did not discover it until the idea had been put into their minds, and thence transferred to their subjects.

Bernheim and Liébeault believe that a real *rappport* exists between the subject and the operator, and that this follows as a natural consequence from the methods employed in inducing hypnosis. Not only does it exist, but, according to Bernheim, the operator's power of evoking hypnotic phenomena depends on it. While Bernheim and Liébeault agree on this point, they differ on another. Thus, Bernheim finds in *rappport* the sole difference between hypnotic and ordinary sleep; while Liébeault, on the contrary, tries by means of it to establish an analogy between them.

My own observations in reference to *rappport* have led me to conclusions similar to those of Braid, viz.: (1) That *rappport* does not appear unless it has been directly or indirectly suggested. (2) That the condition is always an apparent and never a real one. It could always be experimentally proved that the subjects actually had been cognisant of what had been said and done by others who had not been placed *en rapport* with them. In those who did not know what was expected of them, and to whom neither direct nor indirect suggestions of *rappport* were made, this condition did not appear. On the contrary, they heard and obeyed anyone who might address them.

Moll, in "Der Rapport in der Hypnose," published in 1892, comes practically to the same conclusion as Braid in regard to *rapport*, viz., that it is caused by direct or indirect suggestions of the operator, or by self-suggestions which result from the subject's conception of the nature of the hypnotic state.

It is true, as Liébeault has pointed out, that *rapport* frequently exists between the sleeping mother and her child, and that she will hear its slightest cry and yet be unconscious of louder sounds. This, however, has no analogy in the hypnotic state. The *untrained* somnambule responds with equal readiness to the voice of anyone, and, if he has been taught only to respond to one voice, he still hears others. Again, the difference between hypnotic and normal sleep is not, as Bernheim says, that *rapport* exists only in the former. On the contrary, we might with justice establish a distinction between hypnotic and normal sleep, on the ground that *rapport* is absent from the former and is a frequently occurring phenomenon in the latter.

Bernheim's Explanation of Hypnotic Amnesia.

According to Bernheim, everyone possess a certain definite amount of nervous force or cerebral activity. During the waking state this is concentrated in the higher nervous centres—the reasoning part of the brain—while in hypnosis it is concentrated in the lower centres—the imaginative or automatic part. All the impressions received during hypnosis, all the phenomena induced—conceptions, movements, sensations, images—owe their origin to this concentrated and accumulated nervous force. When the subject awakes and resumes his self-control, however, the nervous activity is again diffused through the higher centres of the brain and to the periphery. The impressions received during sleep fade, because, having been perceived, as it were, if the simile may be permitted, by a quantity of nervous light, they are no longer bright enough to be conscious when this light ceases to be concentrated upon them. When hyp-

nosis is again induced, the former state of concentration reappears, and, at the same time, the lost memories are revived.

This explanation is ingenious, but unfortunately is not in accordance with observed facts. If I suggest to a subject that on awaking he will remember the events of hypnosis, he invariably does so. Yet, according to this theory, the redistribution of nervous force to the higher centres should have inevitably prevented this. Again, suggestion may rob the hypnotised subject of the power of recalling the events of previous hypnoses. But, if Bernheim's explanation be correct, the lost memories could not escape revival, seeing that they must have reappeared when the nervous force was again concentrated in the lower centres.

The Influence of the Operator in inducing the so-called Automatism.

In the estimation of the part played by the operator, Braid differs from certain members of the Nancy School. According to the former, the operator merely acts as an engineer who directs the forces in the subject's own body; but the phenomena of hypnosis can also be evoked by ideas previously existing in the subject's mind. The latter regard the operator's *rôle* as a much more important one; not only are his suggestions a mighty force in themselves, but their power is increased by constant undermining of the volition.

For the successful manifestation of hypnotic phenomena, Forel considers it essential that the subject should be under the dominion of the operator, and have lost his own power of concentration and attention. He regards the condition as a battle between operator and subject, in which the former, after capturing outpost after outpost, at last reigns supreme in the central citadel itself. "The mind of a man, A., imposes itself," he says, "upon the mind of another man, B., takes possession of it by entering through some crevice in its armour, and finishes by reigning there more or less as master, and by employing the brain of B. as its docile instrument."

According to Bernheim, the natural tendency that exists in everyone to accept suggestions is gradually and skilfully developed by the operator.

In opposition to the views of Bernheim, &c., I would draw your attention to two classes of cases.

(1) Where the operator has deliberately tried to minimise his own importance in reference to the induction of hypnotic phenomena.

Although I soon ceased to believe that the subject's volition was dominated by that of the operator, I still found, as the result of sensational writings on the question, that a considerable number of my patients objected to be hypnotised, on the ground that it would interfere with their volition. To obviate this difficulty, I changed my method of inducing and managing the hypnotic state. I commenced by informing every new patient that I did not believe it possible for the operator to dominate the volition of the subject, and that, even if such a thing were possible, it could certainly be prevented by suggestion. I explained to my patients that nothing would be suggested without their consent having been previously obtained in the normal state. Under these circumstances, if the suggestions were successful, this would not imply any interference with volition, seeing that their consent had already been obtained. I pointed out that the fulfilment of a hypnotic suggestion frequently demonstrated an increased, not diminished, power of volition. For example, a patient who desired to resist a morbid impulse, but was unable to do so by the exercise of his normal volition, might gain this power by hypnotic suggestion. Thus, the suggestion did not suspend the volition of the subject, but removed the obstacle which prevented the wish being carried into action. Further, as resistance was manifested despite suggested obedience, it was reasonable to expect that this might be enormously increased by training. I suggested, therefore, to all patients during hypnosis, that they should invariably possess this power of resistance, and also, that neither I nor anyone else should ever be able to reinduce hypnosis without their express consent. This change of method did not affect the results. Notwith-

standing the fact that the patients were convinced, and justly so, that they possessed complete control of the whole condition, hypnosis was evoked as easily as formerly, and as wide a range of phenomena was induced.

(2) Where an attempt has been made to teach the subject to evoke hypnosis and its phenomena without the intervention of the operator.

Some six years ago I commenced to instruct patients to hypnotise themselves. This was done by suggesting in hypnosis that they should be able to reinduce the state at a given signal; as for example, by counting "One, two, three." These subjects could afterwards evoke the condition at will. I also found that the use of suggestion during hypnosis was not necessary for the induction of its phenomena. On the contrary, the suggestions could be made equally well beforehand in the waking state. The subject was able to suggest to himself when hypnosis should appear and terminate, and also the phenomena which he wished to obtain during and after it. This training was at first a limited one; the patients, for example, were instructed how to get sleep at night, or relief from pain. They did not, however, always confine themselves to my suggestions, but originated others, and widely varying ones, regarding their health, comfort, or work. Some, trained in this way six years ago, still retain the power of hypnotising themselves.

In such cases it would be difficult, I think, to explain hypnotic phenomena as the result of arrested, or weakened volition; and of outside interference by the operator. It might be objected, perhaps, that the influence of the operator had not been entirely eliminated, on the ground that he had been associated with the induction of the primary hypnosis. The conditions, however, which are more or less frequently associated with the origin of a particular state are by no means essential for its after-manifestation. For instance, the art of swimming is usually taught either by means of a life-belt, or by attaching the pupil to a cord which the teacher holds and guides by means of a rod. These artificial aids, however, are not essential to the art of swimming; they are only useful in its acquirement. It

would be illogical to ascribe a champion's power of winning a race to the presence of a life-belt which he discarded years before. In the same way, it would be unjustifiable to attribute a subject's power of influencing forces within his own body by suggestions arising in his own mind to the influence of the operator who had formerly instructed him how to evoke and direct this power.

Monoideism.

Although Braid and Bernheim differ on many points, they are in complete agreement as to the main factor in the problem. According to both, the essential condition is one of *monoideism*. The mind of the subject is concentrated on a single idea. Only one function is active at any one time; and intensely so, because all the attention is given to it. Other functions are inactive, other sensations unperceived, because the subject has no attention left to give to them. Bernheim, as we have seen, stated that, while the attention might be directed from one point to another, concentration remained. This was regarded as essential and characteristic; the existence and explanation of hypnotic phenomena depended on it. Impressions which under ordinary circumstances would reach consciousness now ceased to do so, not only because they did not happen to be attended to, but also because the subject had nothing left wherewith to attend to them. Thus this is not only a "concentration of attention" theory, but a "concentration and limited quantity of attention" theory.

Beaunis, while admitting the influence of attention in the production of the phenomena of hypnotism, does not believe that all the facts can be explained by the "concentration of attention" theory. "If," he says, "a hallucination is suggested to a subject and realised on awaking immediately afterwards, it might be possible to explain this on the assumption that the attention was still concentrated upon the suggested idea." He does not believe, however, that, when the appearance of the phenomenon has been delayed for a lengthened period, it can be explained in the same way;

for he does not consider it possible that the subject's attention can have been concentrated on the suggested idea all the time.

Gurney, who also rejected the "concentration of attention" theory said: "The energy of attention is not a fixed quantity, bound to be always in operation in one direction or another; nor does the human mind, any more truly than Nature, abhor a vacuum. What do we gain, then, by employing a general term to describe such special effects? When once the [gas] chandelier metaphor is abandoned—when once it is recognised that in a multitude of cases the quantity of attention turned on in one direction is in no way connected with the withdrawal from any other—the idea of a common psychic factor seems out of place and misleading."

This theoretical objection is in accordance with observed facts. Doubtless certain hypnotic states exist in which all the attention, so far as it is called into action, is concentrated upon one idea. In order to prove, however, that directing the attention upon a fresh point necessarily withdraws it entirely from another, it must be shown that the phenomenon which resulted from the first concentration inevitably ceased when the second one arose. A cutaneous analgesia of the arm might, with some show of reason, be said to result from attention directed to the muscles during suggested catalepsy, on the ground that no attention was left wherewith to attend to painful sensations. But while the catalepsy still exists, how, on this theory, can one explain, for example, a cutaneous tactile hyperæsthesia of the same limb, by means of which the subject can distinguish the two points of the compass at half the normal distance? If the subject is unconscious of painful sensations, because his attention is entirely concentrated on his muscular condition, this same lack of attention to the skin ought not only to have prevented abnormal distinctness of tactile impressions, but also to have inhibited the usual ones. The experiment can be still further complicated, for, while still permitting the catalepsy to persist, the cutaneous tactile hyperæsthesia can be associated by suggestion with a cutaneous analgesia over the same area. Now the subject's whole attention cannot

be directed to maintaining a condition of muscular rigidity, if he has still enough of it left to suffice, not only for the increased perception of certain tactile sensations, but also for the selection and inhibition of other painful ones. Further, the opposite of these phenomena can be simultaneously evoked on the other side of the body: the patient's muscles can be paralysed by suggestion, his tactile sensibility abolished, and his sensibility to pain increased. The attention is now directed to six different points, and could, with equal ease, be simultaneously directed to many others. A psychic blindness, for example, could be suggested on one side; a psychic deafness on the other; hyperæsthesia of the sense of smell and taste on one side, and diminished or abolished sensibility on the other, &c., &c. But this is not all, for while the attention is presumably turned in all these different directions, the subject may be engaged in the successful solution of some intellectual problem. A still further complication is possible. Let us suppose, as in the case of one of my own patients, that a fortnight before, in a previous hypnosis, a suggestion to record the time at the expiration of 20,213 minutes, or some such complicated number, had been made; this will be carried out, despite the existence of the various muscular and sensorial conditions already referred to, and the fact that, at the moment of its fulfilment, the patient is engaged in some other mental effort.

This picture of the hypnotic state is neither fanciful nor dependent solely on my own personal observation. The fact that numerous and varied hypnotic phenomena can be simultaneously evoked in the same subject has been repeatedly observed and recorded by others, and, strange to say, even by those who attempt to explain hypnosis by the concentration of the attention upon a single point. It is solely the importance of these facts with regard to this particular theory which has hitherto been so largely overlooked.

Granting that hypnotic phenomena are the result of changes in the attention, one is forced to conclude that these are the exact reverse of those stated by Bernheim as explanatory of the hypnotic state. The simultaneous presence of many phenomena clearly shows that hypnosis cannot be

explained by the concentration of the attention on any one given point. Again, the fact that the multiple phenomena are sometimes similar in character to the isolated ones indicates that the explanation of hypnotic phenomena by means of the amount of the attention concentrated is also fallacious. If *all* the attention is requisite for the production of one phenomenon, and, while this one still lasts, many other hypnotic phenomena are simultaneously induced, whence do the secondary ones derive that excessive amount of attention which is said to be necessary for the induction of the primary one? The hypnotic condition differs, then, from the normal, not because only one phenomenon can be manifested in it at once, but because it may present simultaneously many and more varied phenomena than can be evoked in the normal state at any one time. In one word, hypnosis is a state of *poly-ideism*, not one of *mono-ideism*.

Moll's Theory.

Dr. Albert Moll, of Berlin, attempts to explain the phenomena of hypnosis by trying, like Bernheim, to find an analogy between them and those of normal life. There are, he says, two cardinal facts that we ought to keep in mind : (1) We are liable to be influenced by the ideas of others, and to accept as true statements which we ourselves have not investigated. (2) When a physiological or psychological effect is expected it has a tendency to appear.

The two facts just cited are sufficient, Moll thinks, to explain many of the phenomena of slight hypnosis. The increased susceptibility to suggestion evinced in this state, as the result of the subject's weakened volition, alone separates it from the ordinary waking condition. Moll does not believe that any analogy exists between slight hypnosis and sleep, for in the former there is neither loss of memory nor alteration in consciousness.

In order to explain the phenomena of deep hypnosis, in addition to the factors just referred to, Moll introduces a third, *i.e.*, a *dream consciousness* similar to that which exists in natural sleep. He thinks the *positive* hallucinations of

deep hypnosis are similar to those that occur in dreams, and that they are caused in the same manner by peripheral or central stimuli. The character of the dream aroused, the nature of the mental picture excited, by the *peripheral* stimulus depends, in sleep as well as in hypnosis, upon the personality of the subject. The dividing line between sleep and hypnosis is merely a quantitative difference in the movements occurring in both: in hypnosis these are easily induced; in sleep they are duller, slower, and rarer.

For the production of *negative* hallucinations, Moll considers that the three following factors are necessary:—

(a) The subject's conviction of the absence of an object or sensory impression.

According to Moll, if, in the waking state, we are convinced of the absence of something actually present, this belief tends to prevent our perceiving it. Thus, he says, if a man is working in some place which is generally quiet, and where he does not expect to hear a noise, he would not notice it if one were made.

In Moll's opinion, the hypnotised subject's conviction of the non-existence of an object or sensory impression arises in the following manner:—The suggested idea cannot be supplanted by a voluntary one, for, owing to the alterations in the attention which result from the methods of the operator, the subject is unable to control the ideas conveyed to him, or to put forward his own. External ideas dominate his consciousness. The conviction of the non-existence of an object arises from the subject's weakened will, and his dependence on the operator. The fact that many motor suggestions have already been made, which the subject has been unable to resist, renders further suggestions easy.

(b) Diversion of attention.

The diversion of the subject's attention follows the conviction of the non-existence of the object; he believes no object is present, and, therefore, ceases to direct his attention to it.

(c) Dream consciousness.

Moll considers that the existence of a dream consciousness is also necessary for the explanation of negative halluci-

nations. In it not only do former memory pictures reappear as hallucinations, but sensory impressions no longer, as in normal life, induce feelings or perceptions.

Hypnotic memory.

Moll explains the sudden and often nearly systematic forgetfulness, in reference to hypnotic states, by means of Max Dessoir's theory of the "Doppel-Ich." He considers that the punctual execution of post-hypnotic commands is only comprehensible if, in addition to the primary consciousness, a secondary one works intelligently in us.

To this theory the following objections might, I think, with justice be raised :—

(1) According to Moll, in light hypnosis there is a slight inhibition of the will, which becomes more profound in deep hypnosis. This point has already been referred to in discussing the so-called automatism and the influence of the operator, and I propose to deal with it further in connection with another theory. Meanwhile, I may state that this inhibition of the will has not been present in cases which I have personally observed. Further, in self-hypnosis, where the influence of the operator has been entirely eliminated, hypnotic phenomena can be readily induced.

(2) The objections which have already been raised to the supposed identity of normal and hypnotic sleep are equally applicable to Moll's dream consciousness theory. Sully's views as to the difference between the movements occurring in these conditions have already been referred to.

(3) I know of no instance in which a dream has spontaneously arisen in the hypnotised subject as the result of a peripheral stimulus. It is true that subjects can be taught to exhibit various hypnotic phenomena in response to peripheral stimuli, but, as Moll himself has pointed out, regarding the movements occurring in Heidenhain's cases, the subjects must first have a clear idea of what is expected of them.

(4) Moll assumes a loss of consciousness in deep hypnosis, on which he largely bases the supposed resemblance between that condition and normal sleep. Various objections to this view have been raised in discussing Heidenhain's theory; the hypnotic state is invariably a conscious one, and if

amnesia follows on awaking, this can be prevented by suggestion.

(5) The supposed analogy drawn from normal life, which Moll selects as an illustration of the way in which negative hallucinations arise, from the conviction of the absence of an object or sensory impression, is a peculiarly unfortunate one. A noise is not the less likely to be heard because of previous quietness; on the contrary, a particular noise would be less likely to be detected if it followed numerous others. Again, the cessation of a habitual noise, which we expect to be continued, does not on that account escape notice. When the screw of a steamboat stops this almost invariably arouses the attention of the passengers, even if they are asleep.

(6) Moll's analogies drawn from normal life to explain hypnotic memory are somewhat strained. For example, in reference to the recollection in hypnosis of the events of previous hypnoses, he cites Max Dessoir's statement that he had heard of one person who once during sleep took up a dream at the point where he had left it off on the former night. Surely this is attempting to explain the little known in terms of the less known.

(7) To Moll's explanation of certain hypnotic phenomena by means of the intelligent action of a secondary consciousness I shall again refer. Meanwhile, I would point out that this does not agree with the "involuntary dream consciousness theory"; the two explanations contradict each other.

The Subliminal Consciousness Theory.

Within recent times another theory has arisen. This, instead of attempting to explain hypnotism by the arrested action of some of the brain centres which subserve normal life, would do so through the arousing of certain powers over which we normally have little or no control. This theory appears under various names, "Double Consciousness," "Das Doppel-Ich," &c., and the principle on which it depends is largely admitted by science. William James, for example, says: "In certain persons, at least, the total

possible consciousness may be split into parts which co-exist, but mutually ignore each other."

The clearest statement of this view is given by F. W. H. Myers: he suggests that the stream of consciousness in which we habitually live is not our only one. Possibly our habitual consciousness may be a mere selection from a multitude of thoughts and sensations,—some at least equally conscious with those we empirically know. No primacy is granted by this theory to the ordinary waking self, except that among potential selves it appears the fittest to meet the needs of common life. As a rule, the waking life is remembered in hypnosis, and the hypnotic life is forgotten in the waking state: this destroys any claim of the primary memory to be the sole memory. The self below the threshold of ordinary consciousness Myers terms the "subliminal consciousness," and the empirical self of common experience the "supraliminal." He holds that to the subliminal consciousness and memory a far wider range, both of physiological and of psychical activity, is open than to the supraliminal. The latter is inevitably limited by the need of concentration upon recollections useful in the struggle for existence; while the former includes much that is too rudimentary to be retained in the supraliminal memory of an organism so advanced as man. The recollection of processes now performed automatically, and needing no supervision, passes out of the supraliminal memory, but may be retained in the subliminal. The subliminal, or hypnotic, self can exercise over the nervous, vaso-motor, and circulatory systems a degree of control unparalleled in waking life.

He suggests that the spectrum of consciousness, as he calls it, is indefinitely extended at both ends in the subliminal self. Beyond its supraliminal physiological limit lie a vast number of complex processes belonging to the body's nutrition and well-being. These our remote ancestors may possibly have been able to modify at will, but to us they seem entirely withdrawn from our sphere of volition. If we wish to alter them we must do so by drugs and medicaments, whether the body to be treated is our own or another's.

At the superior, or psychical, end the subliminal memory

includes an unknown category of impressions which the supraliminal consciousness is incapable of receiving in any direct fashion, and which it must cognise, if at all, in the shape of messages from the subliminal consciousness.

Myers arranges hypnotic phenomena into three divisions :—

(1) The great *dissociative* triumph of hypnotism, namely, the inhibition of pain under conditions of nerve and tissue with which it is usually inevitably connected.

Here, psychologically, the whole interest lies in the question whether pain is suppressed together with sensations of every kind, or whether other sensations persist, pain alone being inhibited. Our ancestors, Myers suggests, had already attained to a rough practical knowledge of this distinction. They knew that if you stunned a man by a blow he would not feel the pain for some time. Also, that if you ran pins into particular parts of a witch's body she, although perfectly awake, and conscious of other sensations, would feel no smart.

The second of these discoveries is the more important. By stunning your enemy you only proved that vital functions could continue unimpaired, notwithstanding that the brain's action was so far disturbed that all consciousness was temporarily abolished. By pricking the witch in her "marks"—now called hysterical analgesic zones or patches—you proved that pain was a dissociable accident of organic injury; that other sensations might persist and only that of pain be in some way inhibited. The insensitiveness to pain which runs wild in hysteria is now being directed into useful channels by "hypnotic suggestion." Some *intelligence* is involved in a suppression thus achieved; for this is obtained, not, as with narcotics, by a general loss of consciousness, but by the selection and inhibition from among all the percipient's possible sensations of disagreeable ones alone. This is not a mere anæsthetisation of some particular group of nerve-endings, such as cocaine produces; it involves the removal also of a number of concomitant feelings of nausea, exhaustion, anxiety, which are not always directly dependent on the principal pain, but need, as it were, to be first sub-

jectively distinguished as disagreeable before they are picked out for inhibition. This freedom from pain is obtained without either deadening or dislocating the general nervous system: with no approach either to coma or to hysteria. The so-called hypnotic trance is not necessary; sometimes the pain can be prevented by "post-hypnotic" suggestion destined to fulfil itself after the awakening; and, if there be trance, this is often no mere lethargy, but a state fully as alert and vivid as ordinary waking life.

Myers argues from this that it is plain that hypnotic analgesia thus induced is by no means a mere ordinary narcotic—a fresh specimen of such methods as are already familiar for checking pain, by arresting all conscious cerebration. It is a new departure: the first successful attempt at dissociating forms of sensation which throughout the known history of the human organism have almost invariably been found to exist together.

(2) The *associative* or *synthetic* triumphs of hypnotism, namely, the production and control of organic processes which no effort of the ordinary man can set going, or in any way influence.

Hypnotic analgesia, Myers says, may be classed with equal justice as a dissociative, or as an associative act. The sensations are severed from the main supraliminal current, and thus far the act is dissociative. The group itself, however, has to be formed, and the more complex it is the more this involves some associative act. Inhibition of all the pain consequent on an operation is in reality a complicated associative process. It involves (*a*) the singling out and fitting together of a great number of sensations which have the one subjective bond of being disagreeable; and (*b*) the inhibition of all of them, which thus leaves the supraliminal consciousness in perfect ease.

In further illustration of the associative powers of hypnotism, Myers refers to alterations in the pulse, the secretions, excretions, &c.; he also cites Delbœuf's case of two symmetrical burns on the same subject, one of which ran the ordinary course of inflammation, while in the other the morbid action was arrested by suggestion.

(3) *The Intellectual or Moral Achievements of Hypnotism.*

These, like the others, are based upon physiological changes, but present problems still more profound. The removal of the craving for alcohol and morphia, the cure of kleptomania, bad temper, excessive indolence, &c., are cited as illustrating the moral and psychological changes which suggestion can effect.

Volition, &c.

According to Myers, the hypnotic subject is not a maimed or stunted normal individual; but one who, while he has gained increased power over his own organism, has not at the same time lost his volition or the mental and moral qualities which formerly distinguished him. He admits that there is some difficulty in explaining hypnotic obedience; but holds that this will be refused when the act suggested is contrary to the subject's moral nature. He believes that a complete comprehension of the suggested act exists in the subliminal strata; and that, when great need arises, the subliminal self will generally avoid compliance—not necessarily by awakening the organism into ordinary life, but by plunging it into a hysterical access, or into a trance so deep that the unwelcome order loses its agitating power. The moral tone of the somnambule is, in Myers' opinion, the precise opposite of the drunken condition. Alcohol, by paralysing first the higher inhibitory centres, makes men boastful, impure, and quarrelsome. Hypnotisation, apparently by a tendency to paralyse lower appetitive centres, produces the contrary effect. The increased refinement and cheerfulness of the developed somnambule is constantly noticed.

The Possible Source or Origin of Hypnotic Control over Intimate Organic Processes.

Myers asks whether we can find anything in our ancestry which suggests to us these internal powers of modifying circulation, quickening cell-proliferation, and altering

trophic processes in unknown ways. He admits that the analogies to which we can appeal are vague and remote, yet he says we can point to the general fact that in man and the higher animals an increase in the power of modifying the action of the organism as a whole has evidently been purchased by a decrease in the power of modifying its internal parts or constituent elements. The self-shaping powers of the amoeba, the self-regenerating powers of the worm or crab, die gradually away into the comparative fixity of the organism of the higher mammalia.

It is possible, he thinks, that this fixity is more apparent than real: "We may regard the human organism as an aggregation of primitive, unicellular organisms, which have divided their functions and complicated their union in response to the demands of the environment, and along such lines of evolution as were possible to the original germ. It is possible, too, that all these processes—beginning with the amoeboid movements of the primitive cell—were accompanied by a capacity of retaining the impress of previous excitations, a rudimentary memory which at first constituted all the consciousness which our lowly ancestors possessed. And further—may we not suggest?—as evolution went on, and more complex operations were developed, while the primitive processes of cell-change became stereotyped by long heredity, the memory which represented these earlier changes sank to a low psychical depth, became subliminal, and could no longer be summoned by voluntary effort into the supraliminal sequence of conscious states. How do we know that any psychical acquisition is ever wholly lost? or even that a memory is the weaker because it has sunk out of voluntary control? It may be possible, by appropriate artifices, to recall primeval memories, and to set in motion any physiological process which could at any moment of our ancestral history have been purposely, however blindly, performed."

Thus, Delboeuf thinks that suggestion is not only capable of inhibiting sensations of pain intimately associated with organic injury, and of modifying or arresting various morbid nervous conditions which arise more or less directly from it;

but is also capable of influencing the organic changes which, under ordinary circumstances, would have resulted from the injury itself. In support of this theory, he cites numerous interesting cases, of which the following are examples:—

One of his subjects had her fingers accidentally severely crushed; suggestion at once stopped the pain, and the healing process was abnormally rapid. Another subject had a considerable portion of her thumb cut off. The following day, after a sleepless and painful night, she was unable to use her hand. Suggestion entirely removed the pain, and the wound healed with unusual rapidity.

On June 15, 1886, a strong young peasant woman was shot in the back with a revolver. All attempts to extract the bullet failed; and, during several days, her recovery seemed more than doubtful. When Delbœuf saw her, on June 26, all immediate danger had disappeared, but she was extremely feeble, and could hardly sit up for half an hour a day in an armchair. She had frequent attacks of shivering, followed by profuse perspiration. She could only take liquid nourishment, and this was rarely retained. She suffered greatly from insomnia, and from persistent pain in the abdomen. The bowels were constipated, and defecation and micturition were painful. The wound was kept open with antiseptic dressings. She was hypnotised on June 26, and the process was repeated the following day. Improvement was immediate and marked; she was able to retain solid food, pain disappeared, and the action of the bladder and bowels became normal. By June 30, she was able to stand and walk, and to do light work, such as sewing or knitting. A few days later, she was again hypnotised, and at once recommenced her ordinary domestic occupations. The wound healed rapidly, and she shortly afterwards took another situation. There was no relapse.

The following is an account of the two symmetrical burns already referred to. The experiment was made by Delbœuf, with the object of further testing the influence of suggestion. Delbœuf first explained to the subject, J., what he wished to do, and obtained her consent. He extended both her arms upon a table, heated red-hot a bar of iron, eight milli-

metres in diameter, and applied it to both of them, taking care that the burns should be identical in duration and extent, while at the same time he suggested that she should feel pain in the left arm alone. The operation was performed at seven o'clock in the evening, and immediately afterwards each arm was covered with a bandage. During the night J. had pain in the left arm, but felt nothing in the right. Next morning Delbœuf removed the bandages; the right arm presented a defined eschar, the exact size of the iron, and without inflammation or redness; on the left was a wound of about three centimetres in diameter with inflamed blisters. Next day the left arm was much worse, and J. complained of acute pain. Delbœuf then hypnotised her, and removed the pain by suggestion. The wound dried and inflammation rapidly disappeared.

In Delbœuf's opinion, the persistent belief that one is suffering from disease may sometimes ultimately cause disease, and, in the same way, the conviction that a morbid condition does not exist may contribute to its disappearance. He considers that the organic changes which follow such an injury as we have just described in the case of J. are not alone due to the injury itself, but are also partly caused by the patient's consciousness of pain. The absence or presence of pain may, to a greater or lesser extent, influence vaso-motor conditions. On the one hand, organic injury, unassociated with pain, may not be followed by congestion, inflammation or suppuration; while in an identical injury, accompanied by pain, these conditions may be present. The consciousness of pain, in addition to being sometimes responsible for morbid changes at the site of injury, may also help to spread them to other parts more or less remote; and thus, when pain is removed or relieved, this really means the disappearance or decrease of one of the factors in the organic malady.

According to Delbœuf, experiments like these lead us to suppose that the action of the moral on the physical may be almost, if not quite, equal to that of the physical on the moral. Thence it follows that the idea of physical mischief may produce mischief; and, on the other hand, that the idea of absence of mischief may bring about, or at least favour, a

cure. How, asks Delbœuf, are we to explain the mechanism of this inverse action of the moral on the physical? The action of organs which are dependent upon the sympathetic system cannot be modified voluntarily by the will; the unstriped muscles, the vaso-motors, the glands, act without the intervention of the cerebral hemispheres. In the lower forms of life the animal was just as conscious of what was taking place in its interior as it was of what was happening at its periphery. With the progress of development, however, its attention would be directed more or less exclusively, on the one hand, to the organs which placed it in direct relationship with the external world, and warned it of the passing of outside events of importance to its existence or well-being; on the other, to the means of attack or defence, which it learnt to use from day to day with greater certainty and vigour. At the same time, the cares of the interior would be got rid of more and more completely, and would be confided to a servant who had been trained to look after them, and whose zeal could be depended upon. In a highly developed animal such as man, the importance of conscious life distracts the attention from the phenomena of vegetative life; the continual obligation to provide for the necessities of existence absorbs the will, while the mechanical regularity with which internal organs act renders conscious attention regarding them unnecessary. In ordinary life our attention is mainly concentrated on the external world, the principal source of our pleasures and our pains; and our will is devoted to perfecting our means of attack and defence. The rapid changes of external phenomena mask the regularity of internal phenomena, which accomplish themselves habitually without our knowledge. The care of the vegetative life has been handed over by the will to nervous mechanisms which have learnt to regulate themselves, and which in general fulfil their task to perfection. Sometimes the machine goes wrong, and intervention becomes desirable. The power which formerly voluntarily regulated it has, however, dropped out of the normal consciousness; and, if we desire to find a substitute for it, we must turn to hypnotism. In the hypnotic state the mind is in part drawn aside from the life of relation, while at

the same time it preserves its activity and power. Voluntary attention can be abstracted from the outer world, and directed with full force upon a single point ; and thus the hypnotic sub-consciousness is able to put in movement machinery which the normal consciousness has long lost sight of and ceased to regulate. It may then be able to act, not only on the reflexes, but on the vaso-motor system, on the unstriated muscles, on the apparatus of secretion, &c. If a contrary opinion has till now prevailed, this is because observation has been exclusively directed to the normal exercise of the will. It can, however, in the hypnotic state, regulate movements which have become irregular and assist in the repair of organic injury. In a word, hypnotism does not depress, but exalts the will, by permitting it to concentrate itself upon the point where disorder is threatened.

Somewhat similar views are also expressed by Professor Beaunis. The cerebral activity, at a given instant, he says, represents a collection of sensations, ideas, and memories. Of these some alone become sufficiently conscious to enable us to perceive them clearly and precisely, while the remainder pass without leaving durable traces. In a series of cerebral acts a certain number of intermediate links frequently escape us, and it is probable that the greater number of mental phenomena take place without our knowledge. Sensations to which we do not pay any attention may nevertheless excite cerebral action, and originate, without our knowledge, ideas and movements of which we become conscious. Our brain acts without our knowledge, with an activity of which we are unable to form an idea, and the facts of consciousness are only feeble fugitives from this mysterious work. Hypnotic phenomena, he thinks, afford examples of this unconscious cerebration.

Can all the effects of hypnotic suggestion be explained, Myers asks, by even the most complete revival of ancestral memories ? Do not the moral or psychological achievements represent a point beyond which such analogies will not carry us ? "These changes must," he says, "of course, rest on a physiological basis ; but that basis implies a well-developed human brain. The knowledge of cortical

centres, which must somewhere exist to make such changes possible, can scarcely have been inherited from pre-human ancestors. Nothing, perhaps, in the whole inquiry is of deeper interest than the possibilities thus dawning upon us of disentangling, from the cerebral labyrinth which represents a man's tastes and character, the special brain processes which stand for some special temptation—say, those which represent the reaction of his organism to alcohol. What is the hidden process that to one patient makes brandy as nauseating as it is to a cat—that in another patient makes the morphia craving as impossible as it is to a rabbit?"

Hysteria, a Disease of the Subliminal Self.

Myers does not consider the subliminal self free from disturbance and disease any more than the supraliminal. Subliminal disturbances, he says, are likely to arise and make themselves felt in the supraliminal being. "How shall we distinguish" he asks, "these subterranean from the superficial storms? How shall we recognise, for instance, a disturbance of the 'hypnotic stratum,'—as we may style for convenience' sake, that group of potential perceptions and reactions which are readily evoked in a suitable subject by the hypnotic trance? It would be absurd to attempt to explain *ignotum per ignotius*, the ætiology of disease by its relation to hypothetical strata of the subliminal self. But one remark I must make, since, crude as it may be, it offers at least a chance of light upon a subject at present hopelessly confused.

"I say, then, that our most plausible conception of a morbid disturbance of the hypnotic self is a derangement of functions or capacities which are habitually observed in the hypnotic state, and in that alone. I should say that the reason for so referring the source of such derangement would be increased if the subject were, when hypnotised, aware of the exciting external cause of such derangements, and capable of modifying them in a way impossible to him in waking life.

"Now it is a striking characteristic of the hypnotic self that it can exercise over the nervous, the vaso-motor, the circulatory systems a degree of control unparalleled in wak-

ing life." . . . "Are we aware in practice of any malady or group of maladies in which these functions, these capacities, are the subject of special disturbances? Are there anæsthesiæ appearing, shifting, and disappearing as rapidly as the suggested anæsthesia of hypnotism? Are there anomalous vaso-motor disturbances which seem to follow the patient's mere caprice?

"The reader will answer with the word *hysteria*. And, meaningless or misleading though that term be, it is in fact our first and obvious reply. Not indeed all, but almost all, the phenomena which can be induced by suggestion in the hypnotic state occur spontaneously in hysterical patients.

"But this will not complete our answer. From the point of view of our present analogy the *differentia* of hysteria will be simply an irrational self-suggestion in regions beyond the power of the waking will—a morbid or uncontrolled functioning of powers over the organism which effect profounder modifications than the empirical self can parallel. Thus the production of patches of anæsthesia or analgesia is a characteristically hysterical symptom, and it implies a power of modifying the sensibility to touch or pain which we cannot imitate under ordinary conditions.

"But when hysteria is thus regarded, it is seen that several other maladies fall under the same category. 'Attaques de sommeil,' 'association-neuroses,' 'Zwangs-Vorstellungen,' and a host of monomanias, show a similarly morbid functioning of precisely that class of powers which hypnotism exhibits to us in harmless or beneficent operation. They are self-suggestions of an irrational and hurtful kind. They are diseases of the hypnotic stratum. Hypnotism is not a morbid state; it is the manifestation of a group of perfectly normal but habitually subjacent powers, whose beneficent operation we see in cures by therapeutic suggestion, whose neutral operation we see in ordinary hypnotic experiment, and whose diseased operation we see in the vast variety of *self-suggestive* maladies.

"I would offer this view to the consideration of those who justly realise the close connection between hypnotism and hysterical phenomena, but mistakenly endeavour

to force all the hypnotic phenomena into the hysterical category.

"M. Babinski, for instance, as we have seen, argues as follows on behalf of the Salpêtrière view that all hypnotic subjects are hysterical. The Nancy subjects, he says, although asserted by Nancy doctors to be non-hysterical, yet show in the hypnotic trance phenomena which we observe elsewhere in hysteria alone. For that reason they must, in fact, be hysterical. This, surely, is to reason in a somewhat obvious circle, and those who, with the great majority of competent judges, are convinced that non-hysterical persons may most assuredly be nevertheless hypnotisable, must seek some other explanation for the similarity of phenomena in the two states. That explanation I have here attempted to give by suggesting that hysteria (and many cognate troubles) should rather be said to fall under hypnotism than hypnotism under hysteria. Those self-suggestive troubles exhibit the disordered working of a stratum of the self which is *per se* as normal and as essential to man's completeness as any other, and which surpasses the superficial stratum in the degree of power which the will informing it can exercise over the organism."

To those who, without stigmatising either hypnotism or subliminal manifestations in general as necessarily *morbid*, are yet disposed to style them *abnormal*, and to regard them as a mere curiosity, which can never be closely wrought with human progress, Myers has a further word to say.

The "normal man" is likely, he thinks, to become as question-begging an individual in physiological as the "natural man" in theological treatises. What is man's nature and what is man's norm? If the question were asked in regard to some lower animal type the answer would be a comparatively easy one. But man's end and aim are not so simple as a rabbit's; he must choose between ideals; he must pursue the higher objects, even to some sacrifice of the lower. Thus we should hesitate to assume that Brigham Young had fulfilled man's end and aim more successfully than Sir Isaac Newton.

Myers suggests that psychological experiment is still at much the same point as was medical experiment in the days of Hippocrates; that though we attempt to describe and analyse the psychical nature with which we have to deal, we have scarcely yet invented any instruments for probing or artifices for modifying it. Not only so, but the very idea of trying to modify our psychical selves by deliberate scientific experiment is as foreign and unacceptable to most men, as the idea of modifying his death-rate by sanitation is to the African savage.

Just as the scientific discoveries of such men as Pasteur and Lister have increased our power of checking or curing disease from the physical side, in a manner and to an extent which was not dreamt of by the earlier physicians, so Myers thinks it is possible that in hypnotism we may find a somewhat similar power of influencing psychical conditions.

After criticising adversely the theories of Mesmer, Heidenhain, Charcot, Bernheim, and others, Myers says:—"It is, therefore, as it seems to me, in a field almost clear of hypothesis that I suggest my own—my view that a stream of consciousness flows on within us, at a level beneath the threshold of ordinary waking life, and that this consciousness embraces unknown powers, of which these hypnotic phenomena give us the first sample."

This theory presents many interesting points for consideration.

(I.) *The Hypnotic Powers and the Conditions more immediately associated with them.*

The special point of interest in the cases cited as illustrating the powers of the hypnotic state is the supposed mental condition of the subject. The immediate origin of hypnotic phenomena depends, according to Myers, upon a *voluntary* alteration in the arrangement of ideas. The introduction of the term *voluntary*, with the recognition that all the subject's attention is not requisite for the production of a solitary hypnotic phenomenon, forms an important distinction between this theory and some of those we have already considered.

With regard to Bernheim's theory, I raised the following, amongst other, objections :—

(a) The condition cannot be called one of monoideism, because many phenomena can be evoked at the same time.

(b) It cannot be explained by the concentration of the attention upon a single point, because, again, many phenomena can be evoked simultaneously.

(c) It is not due to arrested, or impaired volition, as we have shown (1) that the subject can resist the suggestions of the operator ; and (2) in cases of self-hypnosis voluntarily create the phenomena for himself.

(d) It could not be explained by suggestion, as this was merely the artifice used to excite the phenomena.

Despite these objections, one must admit that certain phenomena described by Myers, and which are cited by others as illustrating the theory of monoideism, possess one important feature in common, namely, a change in the arrangement of ideas. If, for example, a hypnotised subject, supposed to be under the influence of his operator, sees a hallucinatory cat, and a self-hypnotised subject successfully suggests one to himself, the phenomenon is practically identical in both instances. The only difference is in the explanation of its origin. Bennett believes that the hypnotised subject sees a hallucinatory cat because the genesis of his ideas is not interfered with, but only their voluntary synthesis. Thus, the operator is able to suggest the remembrance of a cat; but the subject, owing to the involuntary arrest of certain mental powers, fails to understand that it is only a remembrance, and believes in its reality. In Myers' opinion, the subject, instead of having lost the power of voluntary synthesis of ideas, has acquired an increased power of voluntary association and dissociation of ideas. The remembrance of the image of a cat has received hallucinatory vividness, not because the subject was unable to check it from lack of voluntary synthesis of ideas, but because he has elected to allow it to become vivid by voluntarily inhibiting the appearance of all the ideas which would have interfered with its clearness, while, at the same time, he has associated with it ideas connected with the remembrance of formerly seen cats.

Bernheim, as we have seen, attempted to explain the phenomena of hypnosis by the *involuntary* concentration of the attention on one point. According to Myers; the mental changes which take place are voluntary ones, and thus, if the phenomena are due to the subject's attention, he must have acquired the power not only of turning it upon one point, but upon several points simultaneously. The inhibition of sensory impressions in hypnosis presents a certain analogy to what is found in the normal state. The student at his books, who wishes to carry on his work without disturbance, may gradually train himself to be unconscious of external sounds. He teaches his attention to concentrate itself upon the problem before him, and to disregard more and more the noises which might distract him. So also, in looking into a microscope with one eye, he may train himself to keep the other eye open, and at the same time to become unconscious of the objects within its field of vision. Here, both the hypnotised and non-hypnotised subject are producing voluntary changes in their attention. Important points of difference, however, exist between the two.

(1) The student does not disregard auditory or visual impressions the first time he tries to do so. On the contrary, prolonged training is often necessary in order to obtain this power; and frequently he is unable to acquire it. In the deeply hypnotised subject, on the other hand, the power can be developed by a single suggestion and with almost absolute certainty.

(2) In the normal subject the inhibition of the sensory impressions is associated with concentration of the attention upon something else. The moment the student closes his books he becomes conscious, for example, of the organ-grinder under his window, whose presence he was ignorant of a moment before. The instant the attention ceases to be directed to the object under the microscope, the brain becomes conscious of the impressions received through the other eye. With the hypnotised subject the condition of the attention is extremely variable. You may have absence of painful sensations at the very moment that you direct the subject's attention to the fact that you are piercing his flesh

deeply with a needle. Again, you may have increased sensory perceptions of the two points of a compass when you have engaged his attention in the attempted solution of a mental problem. This inhibition of sensory impressions by the normal subject might, perhaps, be justly regarded as an acquired automatic act, but it is an automatism which only acts properly when the attention is voluntarily concentrated on something else. At first the attention must have been consciously divided between the problem contained in the book, and the process of inhibiting the sensory impressions. In the hypnotised subject the inhibitory act, seeing that it was performed without previous training, cannot be regarded as completely analagous to the acquired automatic inhibitory act of the normal subject. It can also be performed without conscious concentration of all the attention in another direction. We must not conclude, however, that the hypnotic act is not associated with a concentration of some of the attention merely because (a) the subject is attending to something else at the same time, and (b) because he is not conscious of attending to the particular act itself. It is possible that the hypnotic act may have been performed by means of a conscious concentration of attention which existed in some lower hypnotic substratum of the personality.

(II.) *Moral and Volitional Conditions.*

The views of Myers practically agree with those I have expressed in discussing the question of so-called automatism. I have not, however, seen subjects try to escape objectionable commands in the manner in which he describes it; though some instances of this kind have been recorded by Professor Beaunis and others. In one of these cases the patient refused to awake, after a disagreeable post-hypnotic suggestion had been given. Another, under similar circumstances, rather than fulfil the suggestion, passed from the alert to the deep stage of hypnosis.

(III.) *Hysteria.*

Myers' theory that hysteria is a disease of the hypnotic substratum is an extremely ingenious one, and is the

only reasonable explanation of the resemblance between certain hypnotic and hysterical phenomena with which I am acquainted. As we have seen, those who believed that hypnosis and hysteria were identical stated that the hysterical alone could be hypnotised. On the other hand, those with wider experience have successfully demonstrated that the hysterical are generally, if not invariably, the most difficult to influence. Of this fact Myers' theory possibly affords an explanation. May not the difficulty of inducing hypnosis in the hysterical—of making one's suggestions find a resting-place in them—be due to the fact that the hypnotic substratum of their personality is already occupied by irrational self-suggestions which their waking will cannot control?

(IV.) *The Evidence for the Existence of a Subliminal Consciousness.*

Many cases of alternating consciousness have been observed in the non-hypnotised subject. As a rule, this has been associated with hysteria or other morbid conditions. An interesting case of this kind was recently recorded in *Brain*, by Lewis C. Bruce, M.D. Not only did this patient manifest two separate and distinct states of consciousness, but the right and left brain alternately exerted a preponderating influence over the motor functions. At one time he was ambidexterous and understood English, at another he was left-handed and spoke Welsh. Sometimes the primary waking state has been morbid, the secondary one comparatively healthy. Of this class, Felida X., so ably described by Dr. Azam, is the familiar example.

The work and writings of Edmund Gurney, A. T. Myers, Frederic Myers, Pierre Janet, William James, and many others, have rendered us familiar with the phenomenon of secondary or multiple consciousness in hypnosis. It can be experimentally demonstrated not only that the hypnotised subject possesses a secondary consciousness, which alternates with his primary one, but also that it is possible for the two to co-exist and to manifest different phenomena simulta-

neously. For example, an individual may have his attention concentrated upon the act of reading aloud from a book with which he was previously unacquainted, and, at the same instant, he may be writing automatically—so far as his primary consciousness is concerned—the result of a problem which was suggested to him in hypnosis the moment before that state was terminated. The primary waking consciousness retains no recollection of the hypnotic suggestion; does not know that the secondary consciousness, after the hypnotic state has been terminated, first solves the problem and then directs the motor acts which record it; and is also unconscious of the motor acts themselves.

In Myers' opinion, the general alternations in memory which occur in connection with hypnosis, afford the strongest evidence in favour of a subliminal or secondary consciousness. As a rule, he says, the hypnotised subject remembers waking life, but, when awake, has usually forgotten the events of the hypnotic trance. This statement cannot be accepted unquestioned. According to Braid, amnesia regarding the events of hypnosis only showed itself in 10 per cent. of those whom he hypnotised; while Schrenck-Notzing's *International Statistics* give 15 per cent. It is to be noted also that this amnesia rarely occurs the first time hypnosis is induced, but, on the contrary, frequently shows itself only after the process has been repeated many times. Under such circumstances it is impossible to be certain that the condition was not artificially created by training, in the same way as analgesia hyperæsthesia and other phenomena of hypnosis. Undoubtedly the operator frequently suggests amnesia; but, even when it is carefully explained that the primary hypnosis is rarely or never followed by it, I almost invariably find that the idea is so deeply rooted in the subject's mind that he suggests it to himself. Again, this amnesia can be prevented by suggestion. However, this objection is of little importance. Your next-door neighbour would still continue to possess his former characteristics, even if you broke down the partition between your house and his, and afforded him free facilities for passing from one to the other.

While we must admit that this general amnesia only occurs in a small percentage of hypnotised subjects, forgetfulness in reference to many of the details of hypnotic life is very common, and apparently affords evidence for the existence of not only one, but of several subconscious states. The subject who on awaking apparently remembers what has happened in hypnosis does not really recollect everything that has occurred. He may remember that the prick of a needle caused no pain, but be quite unable to recall the mental phenomena which, in Myers' theory, must have been involved in the selection and inhibition of painful sensations. The memory of this selective and inhibitory process is lost not only to the primary consciousness, but apparently to the ordinary hypnotic one also, for questioning in hypnosis fails to revive it. Again, the patient who wrongly calculated in hypnosis when a complicated time-suggestion would fall due, but successfully carried out the suggestion in the waking state, was unable to recall in hypnosis that she had corrected the original erroneous calculation, while at the same time no recollection either of the original suggestion, or of the erroneous calculation and its subsequent correction, existed in the normal waking consciousness. This forgetfulness, we see, is not only manifested in reference to acts which might possibly be regarded as automatic ones inherited from some ancestral type, but is also shown in regard to others of an entirely different nature, and which could not possibly have arisen in this way. Some of these phenomena, then, cannot be regarded as hereditary automatic acts of the hypnotic state, nor as analogous to normal automatic ones, seeing that they had not previously been performed in the hypnotic condition. The simultaneous appearance in hypnosis of a greater number of phenomena (regarding them from their physical side only) than can be manifested in waking life, may possibly be explained by the existence of several subconscious states. The normal attention, apparently, cannot attend to so many things at once as the hypnotic. Now, as we have seen, certain of these hypnotic acts, seemingly performed unconsciously, really demand intelligent attention; not only did they require it in the past, but, as they are neither inherited nor acquired

automatic acts, they obviously require it now. The evidence in favour of several unconscious states is further strengthened by the fact that these intelligent hypnotic acts to which we have just referred are sometimes performed without any feeling of effort. But, as Ribot says: "Everyone knows by experience that voluntary attention is always accompanied by a feeling of effort, which bears a direct proportion to the duration of the state and the difficulty of maintaining it."

Granting that one or more subconscious states exist in the human personality, and that hypnotic phenomena owe their origin to the fact that we have by some means or other succeeded in tapping them, two questions still remain:—

Thus, let it be supposed that I possess a friend called Brown, who is usually, physically and mentally, an ordinary individual; from time to time, however, he manifests an extraordinary increase of physical power. Again, though still more rarely, he displays a range of mental powers of which he had formerly given no indication. I ask for an explanation: I am told that Brown, as I know him best, is indeed Brown; but that his increased physical powers are due to the fact that when he shows them he is Jones, and his increased mental ones to the further fact that he is then Robinson. Granting that the phenomena afford evidence of three separate personalities, I cannot accept this explanation as a solution of the problem in its entirety. I want to know, first, how did Jones and Robinson acquire their powers, and, secondly, what has been done to or by Brown which has enabled these powers to be evoked.

(1) *How did Jones and Robinson gain their Powers?*

Myers' explanation, or at all events part of it, is that these powers are a revival of those formerly possessed by some lower ancestral type. He frankly admits, however, that the analogies to which we can appeal are certainly vague and remote, and that to find them we must leave the higher mammalia and descend to the crab, worm or amoeba.

Is it reasonable to suppose that the Hypnotic Powers, regarded as a Whole, existed in some lower Ancestral Type ?

Granting that a limited analogy exists between lower animal types and hypnotised subjects as to their power of influencing certain physical conditions, it would, I think, be impossible to establish an analogy between the mental and moral powers of the latter and those of the savage or lower animal. For example, one of my patients, as we have seen, suddenly developed arithmetical powers far exceeding those she possessed in the normal state. She is not likely to have derived them from some savage ancestor who was unable to count beyond five, or from some lower animal, presumably ignorant of arithmetic. Again, the same patient spontaneously solved, in hypnosis, a difficult problem in dressmaking. The power of correctly designing a garment, in accordance with the passing fashion of the present day, can hardly have been derived from some woad-stained ancestor, or lower animal form. Further, the increased modesty of the hypnotised subject, his greater power of controlling or checking morbid passions or cravings, does not find its counterpart in the savage or ape.

Myers admits that the argument from analogy is weakest when we consider the mental and moral powers of hypnosis. But if it is the essential characteristic of the subliminal state that the spectrum of consciousness is extended at both its physiological and psychological ends ; surely an explanation of both extensions is equally necessary. A theory in itself imperfect becomes still more imperfect when every fact that is supposed to establish the extension of one end of the spectrum renders the extension of the other end still more difficult of explanation.

If we admit that Hypnotic Powers are derived from some lowly, non-human Type, is their easy Recovery probable ?

I have seen cases in which all the phenomena characteristic of deepest hypnosis could be readily evoked, absolutely without training, within a couple of minutes of the commencement of the process employed for the induction of the

primary hypnosis. If any of these were derived from amœba, worm, or crab, the rapidity with which they were aroused was surely surprising.

Is it likely that the Powers should have been lost in Development?

Some of the powers of the hypnotic state are said to have dropped out of the supraliminal consciousness in the process of evolution, as their association with it had become unnecessary in the struggle for existence. It must be noticed, however, that many of these powers have not only ceased to be employed automatically or unconsciously, but have also sometimes apparently disappeared altogether; and, until hypnosis was induced, no means existed by which the supraliminal consciousness could evoke them. Now the powers which the hypnotic self possesses are so numerous, varied, and frequently so essential for the comfort or well-being of the individual, that one with difficulty concludes that development is responsible for their loss. Take, for example, the power of inhibiting pain. Granting that some lower type possessed it—a fact difficult to prove—when and why has this important power been dropped? In this over-civilised age, we appear to have abandoned some of the powers of the subliminal self just at the very moment when we most require them, as is shown by the complaints about street-noises and the manners of children. In many instances, at all events, the supraliminal self is sadly embarrassed by the fact that it cannot perform that feat, so easy to the subliminal one, of shutting out undesired sensations of sound; and, in consequence, can neither work by day nor sleep by night.

(2) *What is the Connection between Hypnotic Methods and the Production of Hypnotic Phenomena?*

To this I think no reasonable answer has been given. Personally, I can see no logical connection between the acts of fixed gazing, of concentration of attention, or of suggested ideas of drowsy states, and the wide and varied phenomena

of hypnosis. Hypnotic phenomena do not appear spontaneously, and some of the methods described must have been employed in each case before primary hypnosis was induced. But I cannot conceive the idea that the methods explain the phenomena.

Suggestion in ordinary Medical Practice and its Relation to Hypnotism.

In the opinion of some authorities suggestion is largely intermixed with medical practice and, in some instances, is the most important factor in its success ; while others hold that this mental influence is similar to, or identical with, hypnotism.

The belief that mental conditions exert an important influence upon physical ones is by no means novel, and was held by Sir Henry Holland and Braid. According to the latter, not only was the action of homœopathic remedies a purely suggestive one, but the mental element associated with the administration of drugs in general had been far too much ignored. He pointed out how the patient's faith in, and expectation of a certain result from, a particular remedy might play a not unimportant part in reference to the supposed action of the drug.

In the opinion of Dr. Waller, it is immediately and specifically important for the physician fully to realise the power of suggestion on mind and body. "The physician," he says, "who knows and trusts his own resources will carry his patient through illnesses, where another man will vacillate and despond, and by his face or manner suggest failure and hasten death." Very similar views were expressed at an earlier date by the late Dr. Oliver Wendell Holmes, who pointed out how the pessimism or optimism of a medical man might gravely affect his success in practice.

According to Dr. Wilks : "To sit down in one's chair daily and write on a piece of paper the name of some drug for every ailment, without exception, which comes under our observation is, in the present state of medicine, an absurdity, and is simply a pandering to human weakness. I do not

say that drugs are not useful in a moral sense. I am merely contending that the method is not scientific, as we usually apply this term. I know of no more successful practitioner than the late Sir William Gull, and his treatment was rational, but he did not credit any particular drug with the properties ascribed to it by the patient. His prescriptions very often consisted of nothing but coloured water." Dr. Wilks describes a "sixpenny doctor" at a cheap dispensary, who saw on an average seventy patients each evening, and whose almost universal medicine was a mixture composed of sulphate of magnesia, burnt sugar, and infusion of quassia. The following were his directions for successful practice: "Always give medicines which produce appreciable effects; then, also, the mixture must taste like medicine, and if it have a bad smell the patient will be better satisfied."

Dr. Wilks states that changes in the pathological views of disease have caused the whole method of its treatment to be altered again and again, and that further chemical knowledge frequently shows that the drugs we employ do not possess the qualities that we have been in the habit of attributing to them.

The late Dr. Hack Tuke, in speaking of Sir Andrew Clarke, said: "His favourite drugs were bi-carbonate of potash and a vegetable bitter, but neither drugs nor diet formed the central factor of his treatment, or explained its success. "Suggestion" lay at the root of it all. The term, however, is too mild unless understood in the technical sense in which it has been employed in recent times. In short, Sir Andrew out-Bernheimed Bernheim; he was, in a word, the most successful hypnotist of his day."

In reference to alleged cures by hypnotism and at Lourdes, Mr. Hart says: "So far as I can see, the balance is in favour of the faith-curer of the chapel and the grotto. The results at least are proportionately as numerous, and they are more rapid. . . . The mesmeriser seeks to dominate his subject, becoming the master of a slave. . . . The faith-curer of the grotto strengthens the weaker individuality. He plays upon the spring of self-suggestion. The patient is told to believe that he will be cured, to wish it

fervently and he shall be cured. So far as he is cured, he returns perhaps a better and a stronger man, and his cure is quite as real, and likely to be quite as lasting, as if he had become the puppet of a hypnotiser."

Admitting that Dr. Wilks' views as to the value of drugs are distinctly pessimistic, we must still grant the importance of mental influences in relation to the treatment of disease. Are we justified in concluding with Hack Tuke that these influences are hypnotic? To this point I have already referred in dealing with Bernheim's theories, and have attempted to show some of the points of contrast between suggestion associated with emotional states and suggestion in hypnosis. The non-hypnotised patient believes that he is being influenced by some external agency. In earlier and more superstitious times the priest or saint was the physician; suggestion was administered in concrete form through the medium of saintly relics, or holy wells, and the cure was ascribed to Divine agency. Sometimes, at a later date, the mesmerist was the physician, and the cure was then said to have been effected by some mysterious force or fluid—odyllic, magnetic, &c. "Faith-Healers" and "Christian Scientists," when, as sometimes happens, they ape the priest and act the mesmerist, would appear to be a mixture of these two classes. Luys' iron crowns and India-rubber dolls, Benedikt's magnets, Harness' electropathic belts, and homœopathic dilutions, in which chemical analysis can find no trace of the original drug, differ little, if at all, from saintly relic or holy well; and although they may have potent results when associated with faith and ignorance, shared alike by physician and patient, advancing knowledge is ever likely to rob them of their powers. Fortunately hypnotic suggestion does not run a similar risk; for not only is Braid's discovery of the subjective origin of the phenomena thoroughly recognised by those who practice hypnotism, but it is also (a point no less important) fully realised by their patients.

Amongst the ignorant and superstitious there is still a tendency to prefer those remedial agents which are more or less closely associated with mystery or occultism. In its extreme form this is shown by pilgrimages to Lourdes and

other shrines, and in a lesser degree by the widespread belief that a bone-setter possesses more skill than an average surgeon, because his powers are a gift uncontaminated by mere scientific training. That Mr. Hart should prefer the treatment of Lourdes to that of Nancy is surely a matter for legitimate surprise. The statement that superstitious belief in miraculous cure, arising in this instance from a hysterical girl's hallucinatory vision of the Virgin, is ennobling, and likely to strengthen the weakened individuality, is a novel one. These superstitious methods of treating disease, of which Mr. Hart apparently approves, are just those which hypnotism condemns and exposes, no matter whether the examples be taken from Lourdes, homœopathic practice, or the magnets of Benedikt.

As to the assertion that the numerical results obtained at Lourdes are equal, if not superior, to hypnotic ones, it would be interesting to know upon what statistics this is founded. For, while hypnotic cases are recorded in the same manner as ordinary medical ones, this cannot be said of the miracle cures of Lourdes; for many of them are rendered valueless as evidence by the fact that no medical examination of the patient had been made immediately before and after the reputed miracle, nor is there any list of the number treated.

An interesting account of an examination of the supposed evidence in favour of superstitious cures is to be found in an article entitled "Mind-cure, Faith-cure, and the Miracles at Lourdes." By A. T. Myers, M.D., F.R.C.P., and F. W. H. Myers. [Proceedings of the Society for Psychological Research. Part XXIV., vol. ix., June 1893, p. 150.]

The British Medical Journal and Hypnotism.

In attempting to give an account of hypnotic theories, one cannot in justice ignore the views expressed in a journal so important as that of the British Medical Association. In it, and in various articles contributed by its Editor to more popular journals, hypnotism has been presented under two very different aspects.

At the Meeting of the British Medical Association, for

example, in 1891, a paper by Voisin on the possibility of suggested crime, was adversely criticised by Benedikt. In reply, Mr. Hart asserted that Benedikt's "criticisms on a paper which professed to give facts, had treated it with contempt, denying the facts without having investigated them, and without bringing facts in support of his own views. It was, of course, exceedingly easy to say that such phenomena as those described must be phantasms of the imagination. It was the first thing that anyone without knowledge or investigation would say." Mr. Hart claimed to have "discovered, by testing the experiments, that the broad statements of Mesmer, Teste and Braid, and that school could easily be verified." . . . "Hypnotism was accepted by all the world, somnambulism was accepted by all the world." As to hypnotic phenomena, Mr. Hart said: "They must see these things, and they could see them—not in the hands of M. Voison alone, but in the hands of men who were close observers, such as Professor Déjerine, who had shown them to him half a dozen times; such as Professor Charcot and his students, men of the closest observation and most extreme scepticism."

Mr. Hart's defence of hypnotism, eloquent and generous though it was, had unfortunately little in the way of fact to support it. The theories of the Charcot School are now regarded as differing little from the fallacies of the mesmerists; both having arisen from an imperfect knowledge of the importance of mental influences in relation to hypnotic experiment.

In his later writings Mr. Hart recants his former views. Thus: "It is not too much to say that the majority of observations of hypnotic phenomena, which we are invited to accept on the authority of men of acknowledged scientific competence and indisputable personal integrity, are vitiated by the fundamental assumption that the subjects are trustworthy—that is, neither deceiving nor self-deceived. This source of fallacy is one to which the scientific experimenter is peculiarly exposed. In investigating the phenomena of hypnotism, scientific phenomena must always be controlled and directed by the practical insight of the man of the

world." . . . "Men of science are not as such particularly well qualified to judge of matters in which the disturbing influence of the personal equation is to be taken into account."

Are Scientific Men peculiarly unfitted for Hypnotic Investigation?

Those who, in the discussion on Voisin's paper, were regarded as "men of the closest observation and most extreme scepticism," have now suddenly become abnormally credulous and easy to dupe. Knowledge of one branch of science does not render its possessor an expert in the methods of investigating widely differing ones; but it is generally conceded that scientific training is requisite for successful scientific research. Moreover, the investigators of the physiological, psychological and therapeutical phenomena of hypnotism are many of them just those who had already distinguished themselves in similar branches of science.

Physiological experiments, conducted in a laboratory with instruments of precision, are equally likely to be accurate whether the subject be hypnotised or not. Extensive therapeutic researches, undertaken in mistrust and doubt by Bernheim, with the assistance of Beaunis, a skilful physiologist, and Liégeois, a sceptical lawyer, are surely as trustworthy as the clinical observations of the ordinary physician. Psychological experiments undoubtedly present greater difficulties, and "amnesia" unfortunately cannot be weighed in a balance or precipitated in a test tube. For the establishment of that, and similar hypnotic phenomena, the experiments must be careful, varied, numerous; surrounded by many test conditions, and associated with a knowledge of the possibility of fallacies. Few, however, are so alive to this as many of the observers who have conducted such investigations.

Are all the Subjects of Hypnotic Research Impostors or Self-deceived?

This question has been answered in the affirmative on the assumption that music-hall performers and the like have alone been investigated.

The material which the hypnotist examines, however, is practically the same as that upon which other medical men found their results; namely, patients. These undoubtedly occasionally practise deceit; but it would be rash to assume that all or nearly all are impostors. Braid's personal friends and relatives were the subjects of many of his researches; Wingfield hypnotised University undergraduates; Heidenhain his own brother and other medical men. My own patients include several medical men and lawyers; while Forel, Braid, and others, have hypnotised themselves.

Are the Majority of Hypnotic Observations vitiated by the fundamental Assumption that the Subjects are trustworthy?

This question must be answered in the negative. Not only is the trustworthiness of the subject rarely assumed, but it is generally recognised that its existence does not preclude error. Braid long ago pointed out that a subject, through the hyperæsthesia of his special senses, might obtain an indication of what the operator desired, and thus, without any intention to deceive, render the experiment valueless. The theory, held by Mr. Hart, that the subject is unconscious, has been the origin of many errors of this kind; and control tests which ignore consciousness are absolutely useless.

Is Hypnotic Practice dangerous and valueless?

As an ordinary narcotic, Mr. Hart contrasts hypnotism with opium and sulphonal, and asserts that it is much more dangerous. He says: "As to the treatment of other diseases, I have, I believe, read all that is to be read on the subject, whether from the school of Nancy, of Paris, or of Vienna, and I have arrived at the conclusion . . . that, for curative purposes, it is very rarely useful, generally altogether useless, and often injurious."

Over a thousand works, and possibly even a larger number of minor articles, have been published on the subject of modern hypnotism. Most of these are written in

foreign languages, including Russian and Swedish. Possibly some of the cases recorded in them might with justice be rejected, because of doubtful or insufficient evidence; others, however, cannot be discarded for this reason. For instance, when Wetterstrand claims to have cured a case of morphinomania, and the patient, a medical man, publishes an independent account of it, it is difficult to admit the existence of error. The conclusions drawn by Forel and myself, after visiting Sweden for the express purpose of studying Wetterstrand's work, are entirely opposed to those put forward by Mr. Hart without personal investigation.

As to the dangers to which Mr. Hart refers, these, as we have already seen, have never been met with by Forel and the majority of those who practise hypnotism. Their existence is assumed from the mistaken theory that the hypnotised subject is an unconscious automaton at the mercy of his operator; but, until the genuine character of music-hall experiments can be proved, we are not justified in drawing conclusions from them, and still less in applying these to ordinary medical work.

Summary.

Mr. Hart and the other writers in the *British Medical Journal* have confined their attention almost exclusively to the historical side of hypnotism; but the various mesmeric fallacies to which they largely refer had already been successfully combated by Braid, and, since his demonstration of the subjective origin of hypnotic phenomena, have ceased to interest scientific men. The untrustworthy nature of Luys' observations was pointed out in 1888 by Dujardin-Beaumetz, and since then they have been considered unworthy of serious notice. The fraudulent character of many music-hall performances is generally recognised, and in several countries they are now forbidden. A substantial subsidy to the Medical Defence Union would do more than any mere writing on the subject to suppress quackery in connection with hypnotism and with medicine in general. The omission in the *British Medical Journal* of any account of the physiological, psychological, and therapeutical obser-

vations on the subject of hypnotism, which have been so largely recorded by many distinguished Continental scientists, is to be regretted.

Benedikt and Hypnotism.

In the *British Medical Journal* great importance is now attached to Benedikt's adverse criticisms of hypnotism; notwithstanding that Mr. Hart, had already characterised his statements as purely personal opinions unsupported by facts. On February 3, and 10, 1894, "Hypnotismus und Suggestion," by Dr. Moriz Benedikt, was favourably reviewed in the *British Medical Journal*, and Benedikt's attack upon the School of Nancy was reproduced. Of this the following is an extract: "Such persons have a 'brand of Cain' by which they can be recognised in the form of a 'solemn narrowness of mind'—priggishness, in fact, if the expression may be allowed . . . It is surprising how many of the fanatical devotees of hypnotism bear upon them this 'brand of Cain' . . . 'His book is philosophical in the best sense; he keeps an open mind for truth wherever it is to be found, but his scientific enthusiasm is always controlled and directed by the practical insight of a man of the world. In a word, he 'sees things as they are,' thus realising Matthew Arnold's ideal of culture."

Amongst those "branded" by Benedikt are to be found some well-known and distinguished members of our profession, including Professor Forel of Zürich.

The following extract, described in the *British Medical Journal* as "a thread of intellectual autobiography," forms an interesting commentary on the conception of ideal culture just referred to. "Krafft-Ebing called me a professor of electro-therapeutics and did not mention that I was also a professor of neuro-pathology, a notoriously internationally known fact. Do I not partly owe my great position in the world to my original researches in psycho-pathology? . . . In my veins flows a wide stream of the blood of Esau and, if this bounds to the heart and brain, there arises enthusiasm for truth and justice, and inspiration to answer for both, even against a world in arms" . . . [I am] "a powerful man of the world, whose tongue can become a sword and his pen a lance in the public literary highway."

"Hypnotismus und Suggestion" is regarded in a very different light in Germany, and is even described as a disgraceful account of a personal quarrel with Krafft-Ebing. For example, Schrenck-Notzing states that "Benedikt has attempted to attack Krafft-Ebing and hypnotism without knowledge of the facts, and without having made himself acquainted with recent hypnotic literature. While he glorifies himself, he personally attacks others in language which recalls the jargon of the most degraded journalism, and which cannot be too energetically described as unworthy of a scientific discussion."

What is the Connection between Hypnotism and the Society for Psychical Research?

An attempt has been made to discredit hypnotism on account of its connection with the Society for Psychical Research. Mr. Hart, for example, classes together "spiritists, the stage hypnotist, the living magnets, the Mahatmas, the belated Psychical Researchers, and the ghost-seers. But they are only," he says, "the stunted remnants, the vestigial and atrophied traces indicating the latter stages of ages of development, in which we have outgrown the period when such follies and fallacies were the almost universal heritage of mankind. . . ." The members of the Society "amuse what they are pleased to call their minds in farcical investigations"; but if they would only employ the "control tests" which he had invented, it would be doubtful whether they would find material sufficiently diverting to enable the Society to exist.

The Society for Psychical Research was established for the purpose of investigating those obscure phenomena which alone, amongst all other natural phenomena, had remained uninvestigated by modern science. It is expressly stated that membership of the Society does not imply the acceptance of any particular explanation of the phenomena investigated, nor any belief as to the operation in the physical world of forces other than those recognised by physical science.

The President of the Society is William Crookes, F.R.S.; while his predecessors were Henry Sidgwick, Balfour Stewart, F.R.S., William James and the Right Hon. A. J. Balfour, F.R.S. Amongst the Vice-Presidents and Members of the Council, for 1896, are to be found :—Professors Henry Sidgwick, Oliver J. Lodge, F.R.S., A. Macalister, M.D., F.R.S., William James, J. J. Thompson, F.R.S., W. F. Barrett, F.R.S.E.; also the Right Hon. A. J. Balfour, F.R.S., the Right Hon. G. W. Balfour, Lord Rayleigh, F.R.S., Walter Leaf, Lit.D., J. Venn, D.Sc., F.R.S., and C. Lockhart Robertson, M.D.

In the list of members appear Professors Ramsay, Beaunis, Bernheim, Bowditch, Stanley Hall, Th. Ribot, Liégeois, Lombroso, Charles Richet, Drs. Max Dessoir, Féré, Liébeault, Schrenck-Notzing, Pierre Janet and many other well-known names.

We are fixing a high standard of modern enlightenment if we regard these gentlemen as the belated remnants of former ages of folly!

Professor Sidgwick, in speaking of the Society, says: "We have continually combated and exposed the frauds of professional mediums, and have never yet published in our *Proceedings* any report in favour of the performances of any of them." The following are examples of this destructive work: (1) A report of an investigation by William Crookes, Victor Horsley, and the late Dr. A. T. Myers, of an alleged supernatural phenomenon, to which spiritualists attached great importance; and the investigators none at all. (2) The complete destruction by Dr. Hodgson of the Theosophical claim to miraculous powers and to the existence of Mahatmas. (3) A series of experiments contrived to illustrate the "Possibilities of Malobservation and Lapse of Memory." These practically refuted the assertion that certain phenomena must be due to spirits, because they could not have been produced by mortals. Mr. S. J. Davey, a member of the Society, since deceased, gave several years to the assiduous practice of certain tricks of sleight-of-hand; these he so successfully supplemented by ingenious psychological artifices as to render them inexplicable. It

would be difficult to find any piece of laboratory work on attention comparable in subtlety and skill with Mr. Davey's demonstrations; while, if we wish to protect ourselves and our fellow-creatures against fraud and imposture, this kind of reply is more effective (although more difficult to obtain) than any vague vituperation can be.

When we recognise that the Society has raised "control experiments" to the level of a fine art, the futility of Mr. Hart's suggestion as to his control tests is obvious.

The explanation of my connection with the Society is a simple and natural one. Shortly after I commenced hypnotic work, the late Dr. A. T. Myers drew my attention to the fact that certain members of the Society were engaged in similar researches; and I found in articles—such as "The Problems of Hypnotism" and "The Subliminal Consciousness," by the late Edmund Gurney and F. W. H. Myers—the only attempt, with which I was acquainted, as far as this country was concerned, to find a scientific explanation of the phenomena of hypnotism. It may be well to mention that both Myers and Gurney had received not only general scientific training, but also a fairly extensive medical one. The latter, when already a Fellow of Trinity College, Cambridge, devoted, though he had no intention of practising, several years to medical study in Cambridge and London; while some of his writings—such as "The Power of Sound"—show that few minds in our time have possessed more scientific acumen than his.

Personally I neither believe in, nor have investigated, spiritists, ghosts, or Mahatmas; but the latter fact renders my opinions regarding them valueless. Mr. Hart's criticisms raise a further question: Do scientific men necessarily identify themselves with, or believe in, all they investigate? This assumption is surely unjustifiable; and to confuse Psychical Researchers with Mahatmas—the judge on the bench, so to say, with the convicted criminal in the dock—shows want of discrimination.

Another question remains: Should the scientific examination of obscure and possibly fraudulent phenomena be condemned? According to Mr. Hart: "With so many serious problems awaiting solution, it is not only deplorable, but in

the highest degree discreditable, that minds made for better things should waste their powers in dabbling with what is simply a despicable and degrading imposture." Now, not only do we owe Braid's discovery of the subjective origin of hypnotic phenomena to the investigation of mesmerism, which he had previously regarded as imposture or self-deception, but science in general has been evolved from the errors and superstitions which preceded its birth. Further, as Dr. Wilks tells us: "Medicine began as a superstition, covered and surmounted by fancies and crude theories. By getting rid of these advance has been made, but the fact is not yet sufficiently realised that many of the old fancies still remain in the profession."

Would it be advisable to place the psychologist, like the physiologist, under Government control? Should investigators be compelled to restrict themselves to certain approved subjects, and be refused permission to walk in any of the by-paths of science, lest they should be accosted by some unrecognised question which solicited investigation? Surely freedom to investigate what one likes, when one likes, and where one likes, is not an unworthy ideal, though possibly it may be difficult of attainment.

Hypnotic Theories.—Summary.

If the theory of subliminal consciousness does not satisfactorily explain all the problems of hypnotism, we are at all events indebted to it for a clearer conception, not only of the condition as a whole, but also of many of its component parts.

The following points in this theory seem most worthy of notice :—

(1) That the essential characteristic of the hypnotic state is the subject's far-reaching power over his own organism.

(2) That volition is increased and the moral standard raised.

(3) That the phenomena of hypnosis arise from, or at all events are intimately connected with, voluntary alterations in the association and dissociation of ideas.

(4) Subliminal or subconscious states are more clearly defined than in previous theories.

Considerable analogy exists between Myers' theory and Braid's later one. With regard to the two first points, their views are identical. Little difference exists between them as to the third. Braid and Bennett both appreciated the important part played by the arrangement of ideas in the production of hypnotic phenomena; but, while the latter held that this was involuntary, the former believed that the volition was unimpaired. The existence of alternating consciousnesses was not only recognised by Braid, but was also regarded by him as explanatory of certain hypnotic phenomena. He did not ascribe to it, however, the same importance that Myers does, nor did he try to find in it a possible solution of all hypnotic problems.

While Myers' theory may with justice be described as such a development of Braid's as might have been expected from the increased knowledge due to further accumulation of facts, some of the other theories already considered tend to revert to "mesmeric" types. The views as to the methods by which hypnotic phenomena are evoked especially show this. The mesmerists attached more importance to the means by which the phenomena were excited than to the subject himself. The "odyllic" force was supposed to exercise its power sometimes without the knowledge of the subject, at others in opposition to his will. Everyone was not susceptible to this influence—those who were being termed "sensitives." The Charcot School hold that some inanimate objects are capable of producing many phenomena practically identical with the "mesmeric." Like the mesmerists, they believe that the influence can be exerted without the subject's knowledge, and also that everyone is not susceptible to it. For the term "sensitive" they merely substitute the word "hysterical." Bernheim's theory resembles that of the mesmerists still more closely. He regards the operator, not the subject, as the more important factor, and believes that a skilful operator can hypnotise at least 90 per cent. of mankind. He holds that in many instances the operator can compel the obedience of the subject, and force him to perform criminal acts. On this point he differs from the Charcot School, and agrees with Mr. Hart and the mes-

merists. The power by which he evokes and also explains the phenomena, called, as we have seen, by the mesmerists "odyllic" force, and by Charcot "hysteria," he terms "suggestion." Braid and Myers regard the operator as the mere starter of the phenomena; his existence, according to Braid, is not even necessary. These observers differ from Bernheim in reference to criminal suggestion and various other points already noticed.

If Braid and Myers have done much towards giving us a clearer idea of the hypnotic state, they have also added to the difficulties of explaining it. A conception of hypnosis which limited its manifestations to simple automatic movements was comparatively easy to explain. The hypnotic subject, who, while he has not lost the physical and mental powers of his waking condition, has acquired new and far-reaching ones, presents a very different problem. But normal life contains many problems, both physiological and psychological, which are yet unsolved, and some—such as the causal connection between mental and physical states—which are apparently insoluble; and while this is so, it would be unreasonable to expect a complete explanation of that still more complex state—the hypnotic. Further observation, however, is always giving us clearer insight, if not into the central problem itself, at all events into the phenomena which characterise it. What increased practical advantage this may give us in curing disease, alleviating pain, or improving moral states, time alone will show. Meanwhile, is it too much to hope that the subject may be approached in a true scientific spirit, and discussed with calmness, courtesy, and dignity?

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J. MILNE BRAMWELL.

ACTION UPON ISOLATED NERVE OF ANÆSTHETICS, SEDATIVES, AND NARCOTICS.

*A Demonstration at a Meeting of the Neurological Society,
June 25, 1896.*

BY AUGUSTUS D. WALLER, M.D., F.R.S.

I.—PROPOSITIONS.

- (1) Anæsthetics, in small quantity, temporarily augment; in large quantity, temporarily or permanently depress or abolish the excitability of isolated nerve.
- (2) Carbon dioxide acts as an anæsthetic upon isolated nerve. Nitrous oxide has no appreciable action.
- (3) An isolated nerve is more safely *anæsthetised* by ether than by chloroform.
- (4) There exists an apparent antagonism between chloroform and carbon dioxide as regards their action upon isolated nerve.
- (5) Bromides and other haloid salts exert a depressant action upon the excitability of isolated nerve. This depression depends upon the salt as a whole, more however upon its basic than upon its acid moiety, *e.g.*, K Br is more depressant than Na Br; Sr Cl₂ is reviving; Sn Br₂ is depressant.
- (6) There is a mutual antagonism between potassium chloride on the one hand, and calcium and strontium salts on the other.
- (7) Among alkaloids and other narcotics, some are depressant, others are inert, as regards an influence upon isolated nerve.
Cateris paribus morphine, strychnine, muscarine, atropine, aconine, are inactive.
Extract of opium, cocaine, aconitine, physostigmine, curarine, gelsemine, veratrine, chloral hydrate, butyl chloral hydrate, are depressant.

II.—EXPERIMENTAL DEMONSTRATION OF

- Proposition (3) Ether compared with chloroform.
Proposition (5) K Br compared with Na Br (in deci-molecular solution).
Proposition (7) An inactive alkaloid (morphine) compared with an active alkaloid (aconitine).

III.—LANTERN DEMONSTRATION

of experimental records in evidence of Propositions 1 to 7.

The regular procedure in taking records of the effects of volatile reagents is as follows :—

The nerve, laid upon exciting and leading off electrodes and enclosed in a moist chamber, is tetanised at regular intervals (once a minute) for one-eighth of a minute. The resulting "current of action" is photographically recorded. Measured quantities of air are driven through the reagent into the moist chamber, by means of a pressure-bottle, while the rhythmic excitation of the nerve is in progress.

THE experimental records to be passed under review have been selected as being illustrative of three groups of reagents, viz., anæsthetics, sedatives, and narcotics.

The first two plates are selected instances of normal runs, the first exhibiting deflections of declining magnitude, the second exhibiting deflections of constant magnitude.

The deflections at the beginning and end of each series are standard deflections, by $\frac{1}{1000}$ volt sent through the nerve and galvanometer, and serving, therefore, to indicate the electromotive value of the negative variations.

Throughout all my earlier experiments I obtained normal records of the first type, more or less pronounced. In later experiments I was usually able to obtain records of the second type. The latter type obviously affords the most favourable condition under which to test the effect of reagents; nevertheless experiments made upon nerves giving declining variations are by no means valueless, as indeed we shall have occasion to see (411).

Clearly the first thing to do with such nerves was to test the action of anæsthetics upon them.

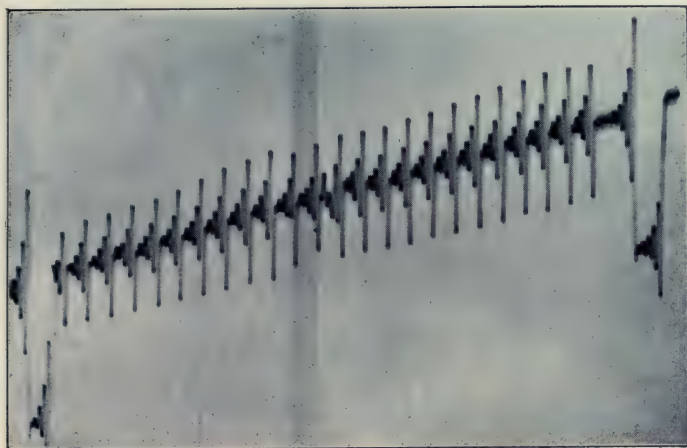
552 is a typical chloroform effect, obtained by driving air into the nerve-chamber, for one minute, through a pair of wash-bottles, the first containing pure chloroform, the second water.

The electrical response of the nerve is finally abolished.

576 is a typical ether effect, obtained by driving air into the nerve-chamber, for one minute, through a pair of wash-bottles; the first containing anæsthetic ether, the second water.

The electrical response of the nerve is temporarily suppressed.

FIG. 1.



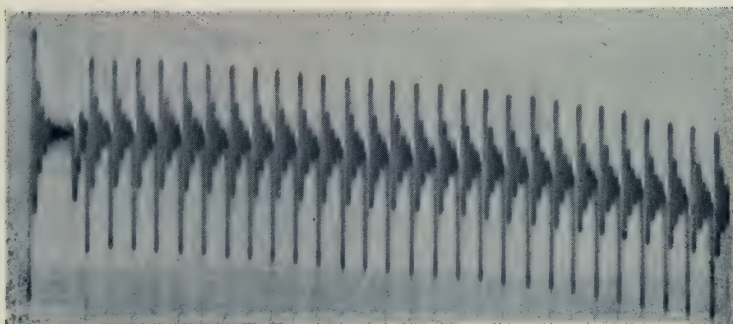
After.

$N_2O.$ ←

Before

Normal run of negative variations of declining magnitude at 1 minute intervals unaffected by gas not acting upon nerve (nitrous oxide). The general decline is due to the falling "current of injury." The decline referred to in the text is of the magnitude of successive deflections.

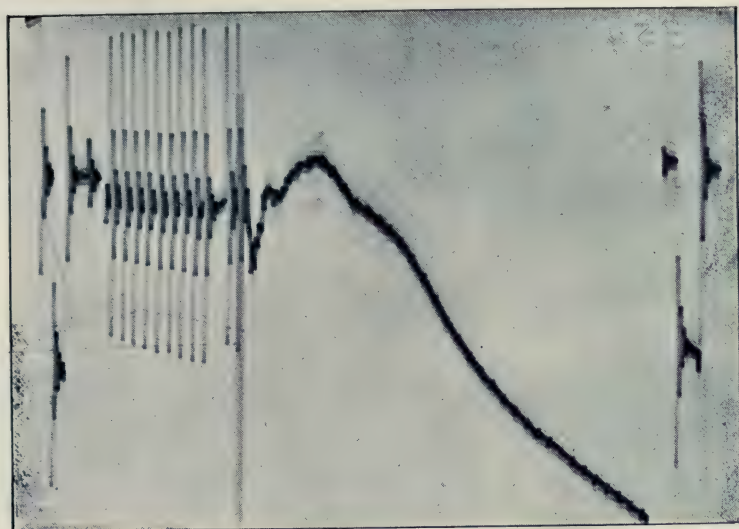
FIG. 2.



→

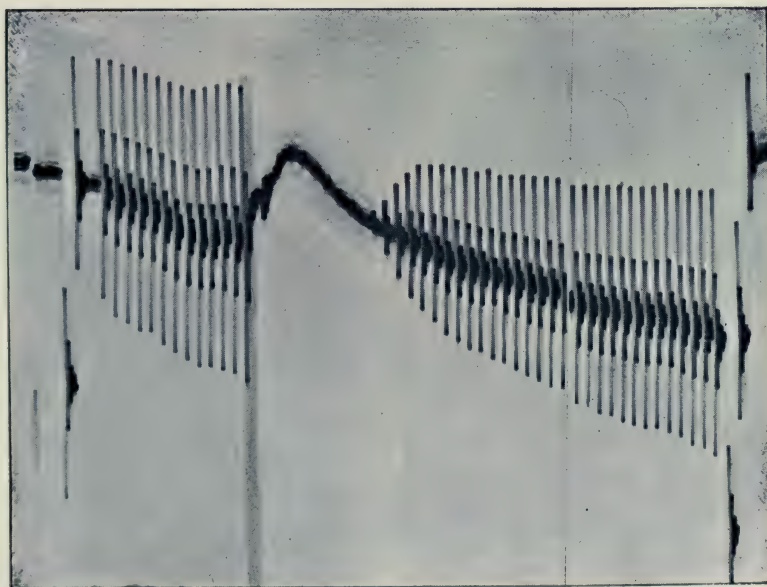
Normal run of negative variations of regular magnitude at 1 minute intervals. The general decline of the base line is not the decline referred to in the text, and is due to the falling "current of injury."

FIG. 3. (552)



Before. ↑ —→
Chloroform. After.

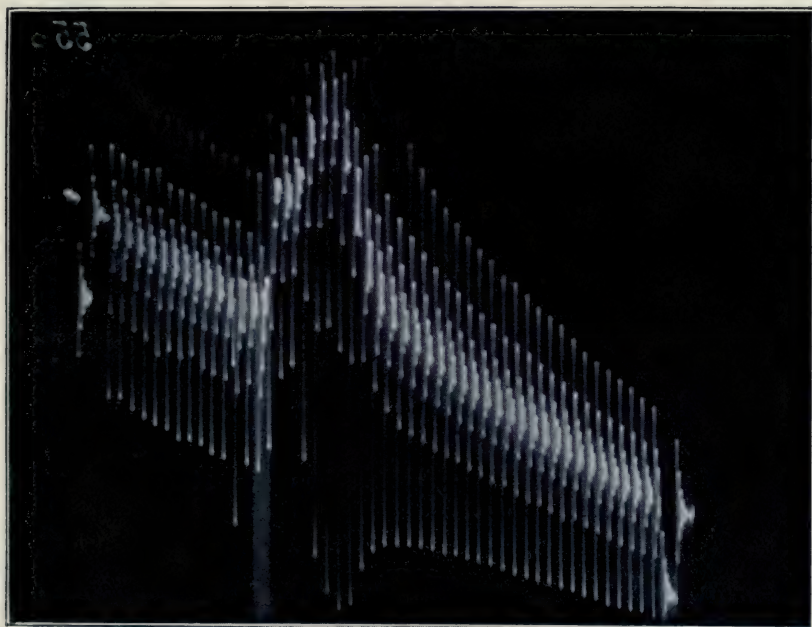
FIG. 4. (576).



Before. Ether After.

512

FIG. 5.
(556) Weak Ether effect.

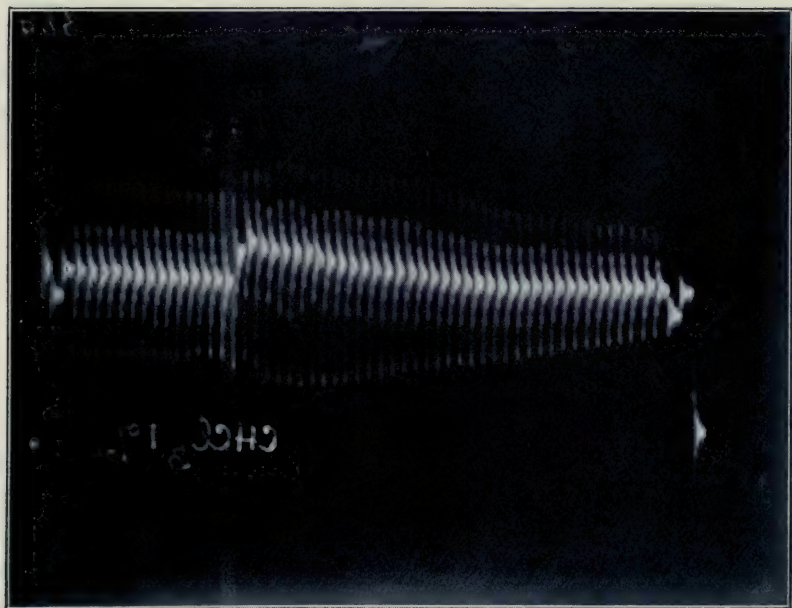


Before.

Et_2O .

After.

FIG. 6.
(669) Weak Chloroform effect.

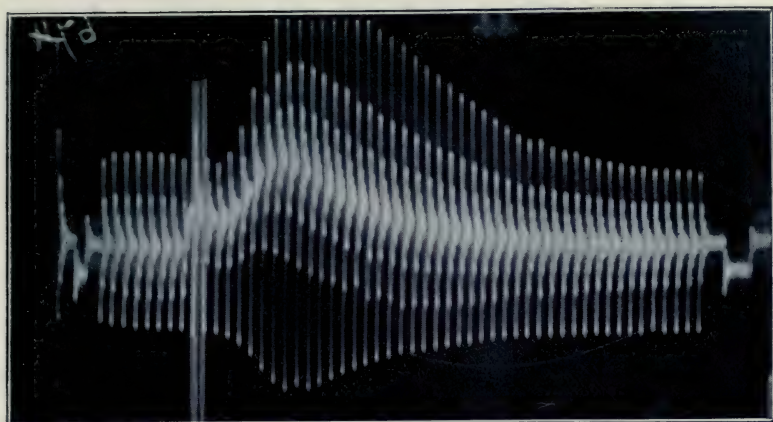


Before.

CHCl_3 .

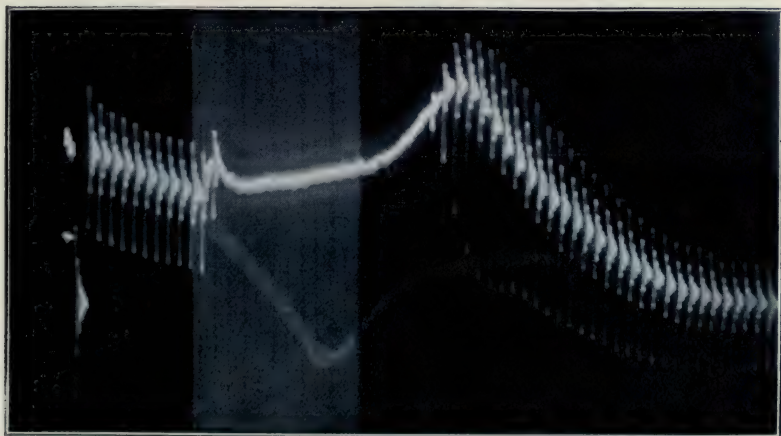
After.

FIG. 7.
(674) Effect of "little" CO_2 .



Before. CO_2 . After.

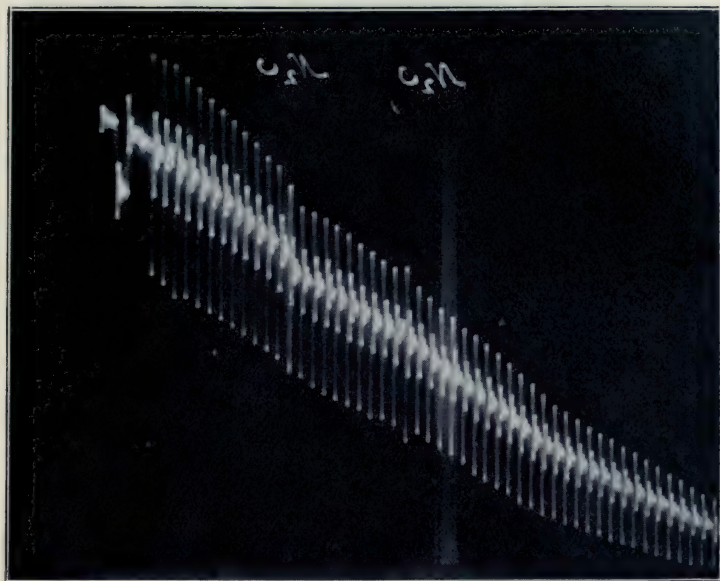
FIG. 8.
(627) Effect of "much" CO_2 .



Before. CO_2 . After.

FIG. 9.

(411) Absence of effect of Nitrous Oxide.



N_2O .

N_2O .

FIG. 10 (described in text).

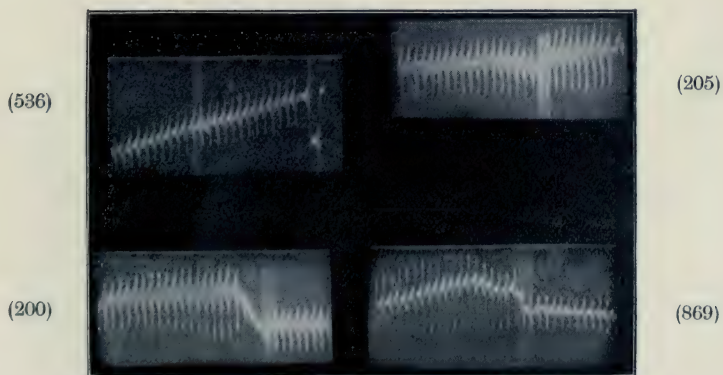


FIG. 11.

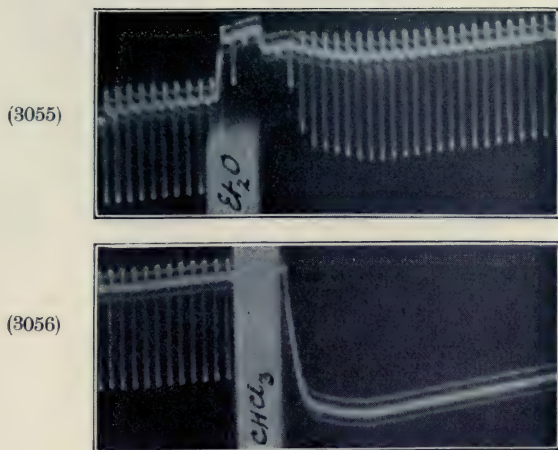


FIG. 12.

Action of Chloroform with and without Carbon Dioxide.

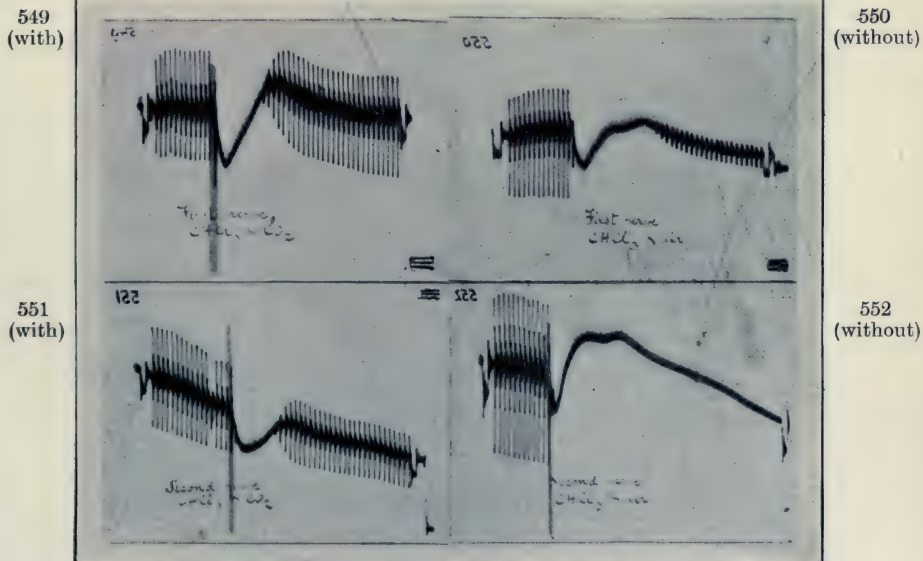
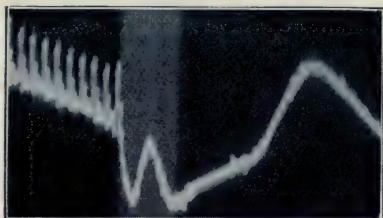


FIG. 13.

Action of Ether and of Chloroform upon retinal excitability by light.

Chloroform.
(3046)



Ether.
(3049)

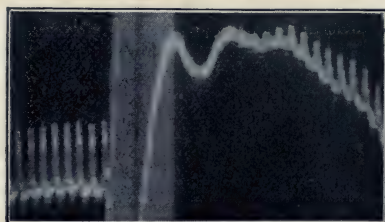
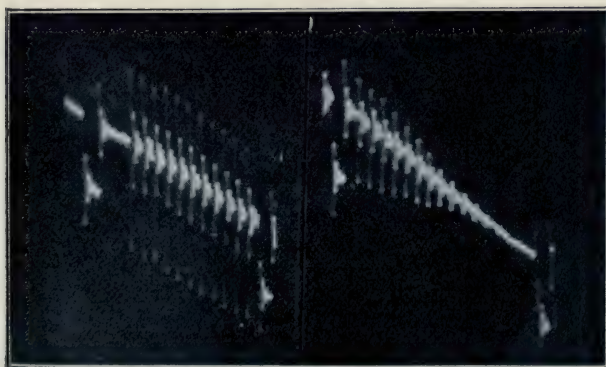


FIG. 14.

(410) Effect of Potassium Bromide.



Before.

↑
KBr. $m/10$

After.

This is a representative contrast, exhibiting one of the most definite practical results of these experiments, and in view of its importance I wish to shew it to you as a going experiment.

EXPERIMENT I.¹

ACTION OF ETHER AND OF CHLOROFORM

(and air saturated with vapour, i.e., with 50 % ether, and 12 % chloroform).

Time. Deflection.			Time. Deflection.	
0 min.	—10·5		16 min.	—14
1 "	—11·5	CH Cl ₃ 750 c.c. of air through wash-bottle.	17 "	—12
2 "	—11·5		18 "	—5
3 "	—11		19 "	—1
4 "	—11		20 "	0
Et ₂ O 1,000 c.c. of air through wash-bottle.	5 "		21 "	0
	6 "		22 "	0
	7 "		23 "	0
	8 "		24 "	0
9 "	0		25 "	0
10 "	0		26 "	0
11 "	—8		27 "	0
12 "	—18		28 "	0
13 "	—17		29 "	0
14 "	—15		30 "	0
15 "	—15		120 "	0

¹ The numbers given in the protocols of experiments I., II. and III., are those actually noted at the meeting, with the exception of those in square brackets which were taken after its conclusion.

In a series of rehearsal experiments made the day before the meeting, the numbers were as follows:—

June 24, 1896. I. *Ether and Chloroform.*

Time. Deflection.			Time. Deflection.	
0 min.	—9		16 min.	—10
1 "	—9	Chloroform: 500 c.c. air through wash-bottle.	17 "	—10
2 "	—9		18 "	—10
3 "	—8		19 "	—8
4 "	—7·5		20 "	—8
5 "	—7·5	Ether: 700 c.c. air through wash-bottle.	21 "	—8
6 "	—7·5		22 "	—6·5
7 "	—7		23 "	—6
8 "	—6		24 "	—2
9 "	—5		25 "	0
10 "	—1		26 "	0
11 "	—1		27 "	0
12 "	—1		28 "	0
13 "	—2		29 "	0
14 "	—8		30 "	0
15 "	—10		90 "	0

II. *Potassium Bromide and Sodium Bromide.*

<i>First nerve.</i>		<i>Second nerve.</i>	
Time.	Deflection.	Time.	Deflection.
1 min.	—11	1 min.	—9
2 „	—11	2 „	—9
3 „	—12	3 „	—9

Bath of KBr, $\frac{m}{5}$ sol. for 10 min.		Bath of NaBr, $\frac{m}{5}$ sol. for 10 min.	
14 „	0	14 „	—9
15 „	0	15 „	—9

Put aside in NaCl, $\frac{m}{10}$ sol. for 2 hours.	
135 „	—6

III. *Morphine and Aconitine* (same nerve as that used for NaBr).

Time.	Deflection.
0 min.	—9
1 „	—9
2 „	—9

Bath of Morph. Hydrochl. $1 \frac{o}{o}$ sol. for 1 min.	
4 „	—12
5 „	—12

Bath of Aconitine Hydrochl. $1 \frac{o}{o}$ sol. for 1 min.	
7 „	—8
8 „	—3
9 „	0
10 „	0

While this experiment is in progress, we may examine the records selected in evidence of Propositions 1 and 2.

If, instead of blowing air through pure chloroform or pure ether in the wash bottle, we send it through chloroform and water, or ether and water, an immediate augmentation of the nerve-response is produced, more easily indeed in the case of ether, which even undiluted sometimes produces this effect.

556. Weak ether effect.

669. Weak chloroform effect.

The primary augmentation is evidently more marked in the case of weak ether than in that of weak chloroform; and another point of difference in my hands has been that a nerve emerging from ether anæsthesia frequently exhibits a

secondary augmentation, which I have never seen in the case of chloroform.

Carbon dioxide, which has sometimes ranked clinically as an anæsthetic, has, in these experiments upon isolated nerve, behaved in a typically anæsthetic manner.

Whereas nitrous oxide, which clinically is admitted to be an anæsthetic, tested under similar conditions, has proved to be totally inert.

The inference is so obvious that I may expressly forbear to draw it, leaving you to judge, as you may think fit, the experimental data in point.

674 illustrates the effect of little CO_2 , primary augmentation.

627 illustrates the effect of much CO_2 , primary abolition, secondary augmentation.

411. The record of a spontaneously declining series, taken at a period when I did not know how to ensure a regular series, shows that N_2O blown into the nerve-chamber upon two occasions has remained without appreciable effect.

These points are summarised in the next group, which includes the action (or rather, want of action) of one other gas, oxygen namely.

205 illustrates the absence of effect of oxygen.

536 illustrates the absence of effect of nitrous oxide.

869 illustrates the effect of little CO_2 .

200 illustrates the effect of much CO_2 .

I should be very much inclined to dwell upon the action of carbon dioxide in more detail; it has proved by far the most interesting gas to study minutely, and has tempted me away from the particular study of anæsthetics to that of the probable production of CO_2 by the nerve itself. I have, however, considered this matter at length elsewhere, and shall therefore only say that the conclusion formulated in Proposition 2 is based upon many hundreds of experiments—upon isolated nerve, at any rate, CO_2 is a typical “anæsthetic.”

Still, in connection with the question of anæsthesia, we may examine three more records—the first two, 3055 and

3056, in reaffirmation of what has already been characterised as a result of practical significance in the ether *v.* chloroform issue; the third, a group of records that I should prefer to characterise as an interrogation, inviting or actually demanding further experimental trial (549, 550, 551, 552). Is CO_2 an antagonist of chloroform, and if so, under what conditions?

3055 exhibits the action of ether upon a "polarisation increment" in nerve.

3056 exhibits the action of chloroform upon the same.

549 exhibits the effect upon a nerve of a mixture of CO_2 and CHCl_3 .

550 exhibits the effect upon the same nerve of a mixture of air and CHCl_3 .

551 and 552 exhibit the same contrast; in the former CO_2 and CHCl_3 , in the latter air and CHCl_3 .

Finally—and because this is an instance upon another organ even more nearly related to the brain than are the nerves—here is a similar comparison between ether and chloroform upon the retina, excited at one minute intervals, by exposure to the light of a standard candle for a period of one-eighth minute at a distance of two feet.

3046. Action of ether upon the retinal current of a frog's eyeball.

3049. Action of chloroform upon the retinal current of a frog's eyeball.

The regular procedure in taking records of the effects of REAGENTS IN SOLUTION is as follows:—

The nerve is excised and put aside in physiological saline. The recording apparatus, exciting apparatus, electrodes, circuit, and keys having been verified, the nerve is laid across the exciting and leading off electrodes in the moist chamber. Its negative variation is then tested. After these preliminaries the recording apparatus is set going, and the standard deflection by 0.001 volt is recorded; the exciting apparatus is then set going, and the negative variation at each minute is recorded for at least ten minutes, affording a normal series of deflections from a nerve taken from physiological saline, *i.e.*, as normal as possible. At the end of this time the nerve is removed from the electrodes, placed in the test solution for one minute, replaced on the electrodes, and the record continued for twenty to sixty minutes. A

deflection by 0.001 volt being taken at beginning and end of the series which exhibits the negative variations of the drugged nerve.

A diminution or augmentation of the deflection might be due to diminution or augmentation of the variation of potential, or to an augmentation or diminution of the resistance of the nerve. The graduation by $\frac{1}{1000}$ volt is intended to reveal any gross alteration of resistance, either by drying in the course of a prolonged experiment, or in consequence of the immersion and readjustment of the nerve when tested by reagents in solution. In the latter case, however, the alteration of resistance is much smaller than might be expected, if each time the nerve is laid across the electrodes it is first laid for a few seconds upon clean white blotting-paper to remove superfluous fluid. If this is neglected the resistance may be considerably diminished by a saline bath, involving an unduly large alteration of the subsequent negative variations—either augmentation by diminution of the general resistance, or diminution by increased derivation of nerve current through surrounding moisture. Even with this precaution there is always some alteration of resistance, not enough to mask the results of ordinary experiments, but sufficient to forbid of the too rigorous measurement of records.

These are some instances of the magnitude of the alteration in earlier and in later experiments. (1) is the deflection by $\frac{1}{1000}$ volt before a bath; (2) immediately after; (3) thirty to forty minutes later.

	(1)	(2)	(3)		(1)	(2)	(3)
271 ..	12	14	13	410 ..	14	15	14
275 ..	13	15	12	471 ..	14	15	14
281 ..	12	17	10	495 ..	30	30	29

In all experiments I attach less importance to the comparison between deflections before and after a bath than to the gradual change, whether diminution or increase, subsequent to the bath, when the nerve is presumably coming more and more under the influence of the solution that has soaked into it.

The salts and other simple reagents are prepared in the form of equimolecular solutions in physiological saline (0.58 per 100). The solutions are generally of deci-molecular strength, *i.e.*, they contain $\frac{1}{10}$ molecular weight grammes of salt in 1000 c.c. H_2O .

Na Br	1.027 grammes in 100 c.c.	Na Cl 0.58 per cent.
K Br	1.188	" " "
NH ₄ Br	0.978	" " "
Na Cl	0.584	" " "
K Cl	0.744	" " "
NH ₄ Cl	0.535	" " "
Na I	1.495	" " "
K I	1.656	" " "
NH ₄ I	1.445	" " "
Li Br	0.967	" " "
Ca Br ₂	2.000	" " "
Au Br ₃	4.370	" " "
Sr Br ₂	2.475	" " "
Sr Cl ₂ · 6 H ₂ O	2.663	" " "

Ca Cl ₂ . 2 H ₂ O	..	1.468	grammes in 100 c.c. Na Cl 0.58 per cent.
Au Cl ₃	..	3.036	„ „ „ „
K HO	..	0.560	„ (by titration)
Na HO	..	0.400	„ „
H Cl	..	0.365	„ „
H Br	..	0.810	„ „

The above has been the usual plan of procedure which, for the purpose of the present demonstration, I must modify somewhat in order to bring the desired comparisons within reasonable limits of time.

I am about to take a comparison between two nerves, immersed for ten minutes in ^M solutions of KBr and NaBr respectively, and by filling in the accompanying table shall expect to illustrate the familiar clinical fact that KBr. is a more powerful sedative than NaBr.

EXPERIMENT II.

POTASSIUM BROMIDE AND SODIUM BROMIDE.

First nerve.

Normal Deflection = 20

Bath of KBr, m/5 solution, for 10 minutes.

Subsequent Deflection = 0

[2 hours later, the nerve = 15 having
been put aside in NaCl, m/10 solution.]

Second nerve.

15

Bath of NaBr, m/5 solution, for 10 minutes.

18

Note.—The deflection by 0.001 volt was 8 to 10.

The KBr nerve now gives no negative variation, the NaBr nerve gives a variation hardly smaller than normal.

The KBr nerve is not dead, its excitability is not definitively abolished; if the nerve is put back in physiological saline, and tested in two or three hours, its excitability would be found to have returned. I shall not be able to show this return on this particular nerve, but inasmuch as the fact must be regarded as being of some importance, I will put upon the electrodes a nerve that was similarly tested by KBr ^M three hours ago. Its normal deflection

before KBr was 12, three hours ago; after KBr it was zero, and now it is 12. The reason for the demonstration of this return is, that it is calculated to convince us that as regards KBr we have had to do with an action that may properly be characterised as sedative, and not merely with a killing of the nerve, such as may be effected by any strong reagent.

Here are records of experiments similar to that of which we are awaiting results, but with rather weaker solutions, viz., KBr $\frac{M}{10}$ (410) as compared with NaBr $\frac{M}{10}$ (272). The difference of effect is obvious, and equally so the inference to be drawn. And here is a third plate (280), showing the effect of KCl, and confirmatory of the obvious inference that the depression by KBr is a potassium rather than a bromide effect.

I have made this comparison a large number of times, under all sorts of conditions, and always with a similar result. Of all the bromides in ordinary use, that of potassium is the most active; that of sodium is the least active. The clinical parallel is obvious, and is further borne out by the effect upon nerve of the bromide of ammonium, which, under similar conditions, has an effect intermediate between those of KBr and of NaBr.

274. Ammonium bromide $\frac{M}{10}$.

One of the most obvious inferences to be drawn from the comparison between KBr and NaBr, is that which is, I believe, accepted by most pharmacologists, viz., that the action of a salt depends upon the basic rather than upon the acid element. And this point has been very clearly borne out by other salts—by the effects of, *e.g.*, potassium iodide and potassium chloride.

It is, I think, unnecessary, and would therefore be tedious, to exhibit the whole series of haloid salts in this way. It will be enough to say in this connection, that while potassium iodide gives a potassium record, sodium iodide gives a sodium record.

I should, however, like to show three later records confirmatory of this capital point, in which a “polarisation

increment" (into the physiological nature of which I will not enter now, but which is on technical grounds a convenient index to take) has been utilised as the physico-physiological sign of nerve activity.

3071. Effect of $\text{NaBr } \frac{\text{M}}{10}$ upon a series of polarisation increments.

3072. Effect of $\text{KBr } \frac{\text{M}}{10}$ upon a series of polarisation increments.

3087. Effect of $\text{KCl } \frac{\text{M}}{10}$ upon a series of polarisation increments.

The tables of measurements, and the curves constructed from them, exhibit sufficiently well the relative efficacy of the nine possible combinations between K, Na, Am, and Cl, Br, I. The numbers must not, however, be taken too strictly, they represent results of single experiments, and it is quite possible that the precise order of efficacy might turn out a little different on a larger number of trials.

The points upon which there can be no doubt are:—

The order of efficacy of the three bases K, Am, Na.

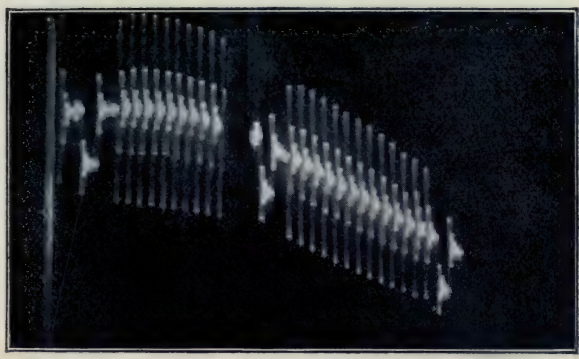
The comparative insignificance of the acid elements, inclusive of the element Br.

But the point upon which there can be doubt is:—

The order of efficacy, if any, of the three acid elements with any one base.

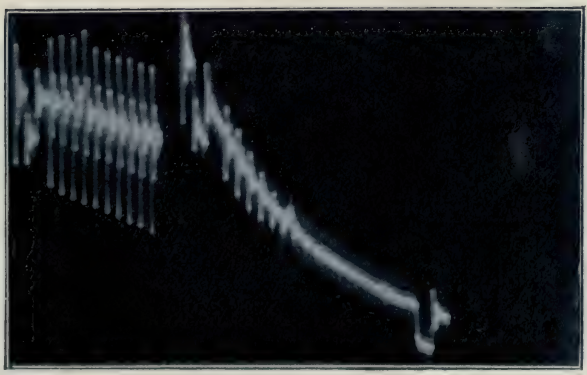
	<i>Chlorides.</i>			<i>Bromides.</i>			<i>Iodides.</i>		
	Am Cl (281)	Na Cl (282)	K Cl (280)	Am Br (274)	Na Br (272)	K Br (271)	Am I (283)	Na I (286)	K I (285)
Norm.	100	100	100	100	100	100	100	100	100
5 min.	76	68	50	75	91	55	75	95	32
10 „	65	55	12	65	82	22	68	95	0
15 „	53	50	0	60	75	0	61	90	0
20 „	41	37	0	50	75	0	48	90	0
25 „	30	27	0	—	—	0	—	—	0

FIG. 15.
(272) Effect of Sodium Bromide.



Before. ↑ After.
 Na Br $m/10$.

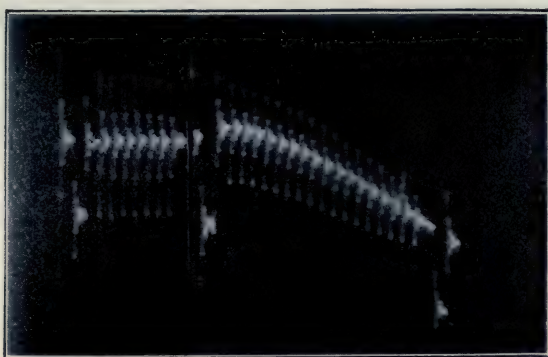
FIG. 16.
(280) Effect of Potassium Chloride.



Before. ↑ After.
 KCl $m/10$.

FIG. 17.

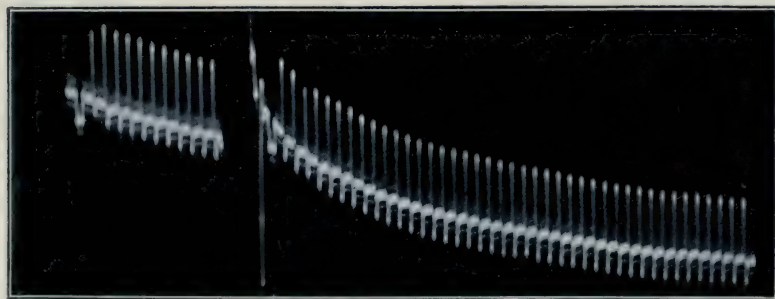
(274) Effect of Ammonium Bromide.



Before. ↑ After.
AmBr $m/10$.

FIG. 18.

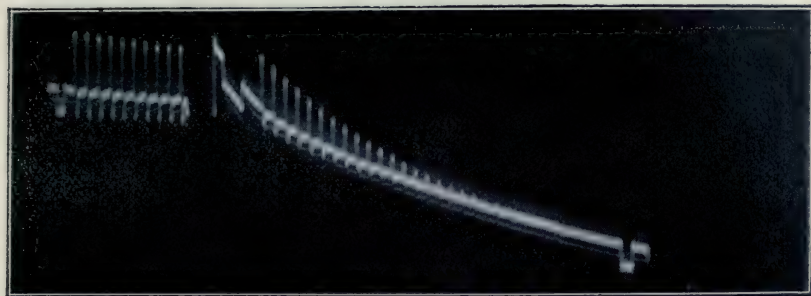
(3071) Effect of Sodium Bromide upon a "series of polarisation increments."



Before. ↑ After.
NaBr $m/10$.

FIG. 19.

(3,072) Effect of Potassium Bromide upon a series of "polarisation increments."



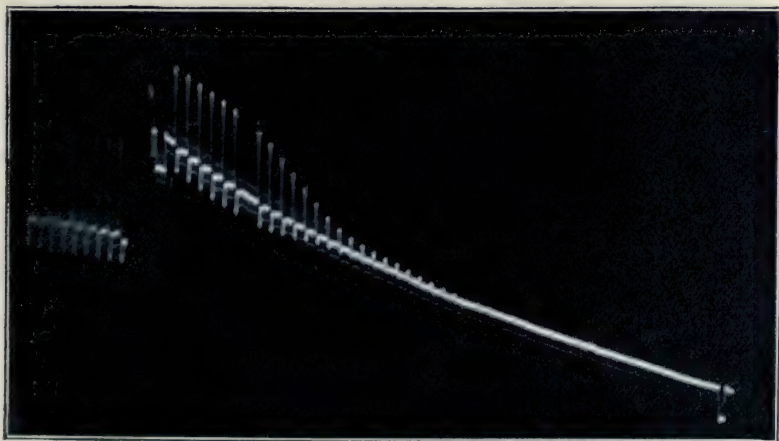
Before.

↑
KBr $m/10$.

After.

FIG. 20.

(3,087) Effect of Potassium Chloride upon a series of "polarisation increments."

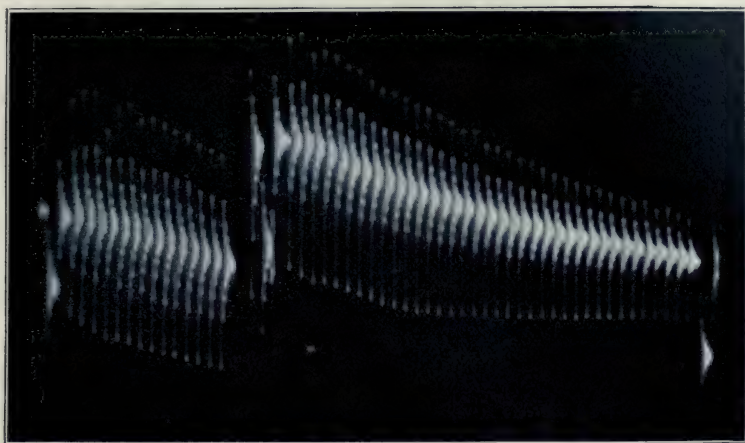


Before.

↑
KCl $m/10$.

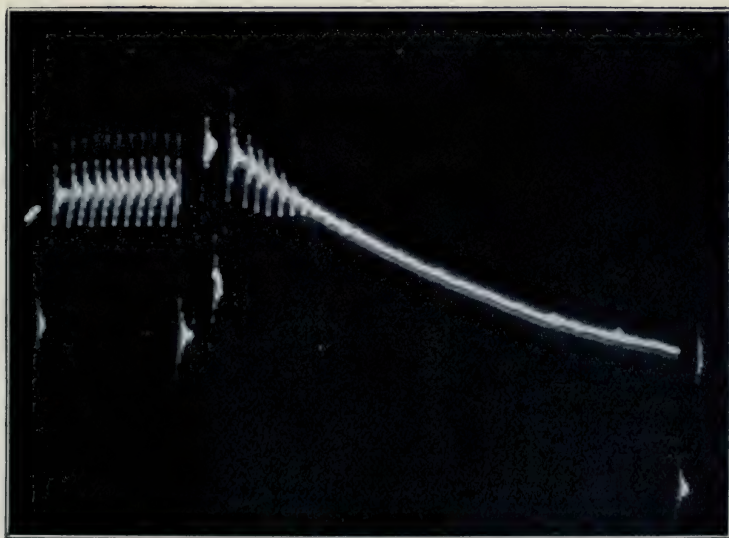
After.

FIG. 21.
(790) Effect of Sodium Hydrate.



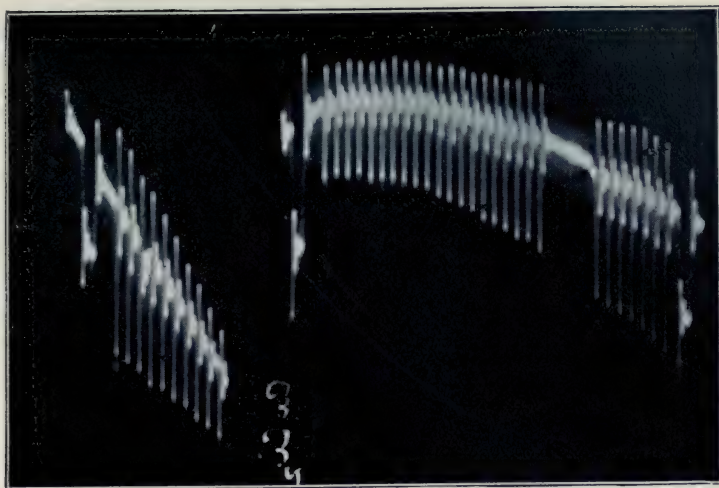
Before. \uparrow After.
NaOH $m/20$.

FIG. 22.
(720) Effect of Potassium Hydrate.



Before. \uparrow After.
KOH $m/40$.

FIG. 23.
(366) Effect of Calcium Chloride.

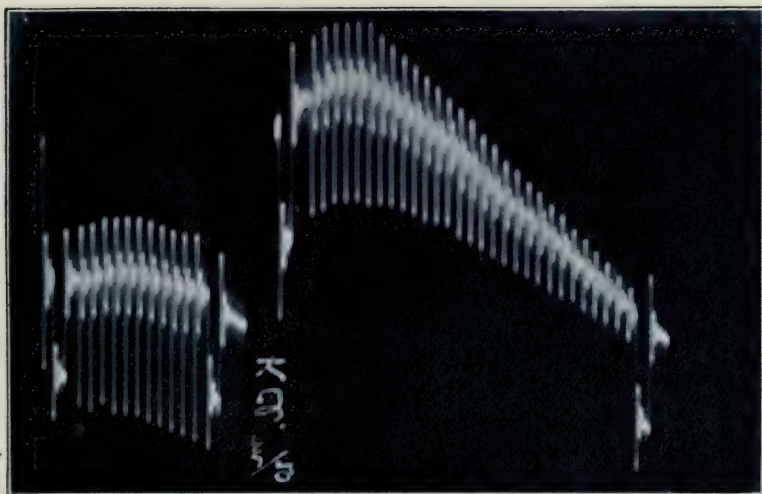


Before.

↑
 $\text{CaCl}_2 \text{ m/10.}$

After.

FIG. 24.
(367) Subsequent effect of Potassium Chloride.



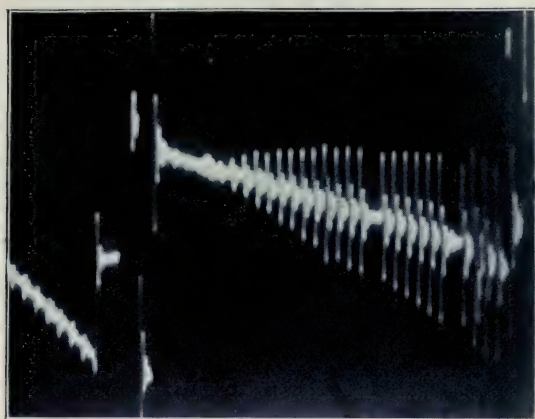
Before.

↑
 KCl m/10.

After.

FIG. 25.

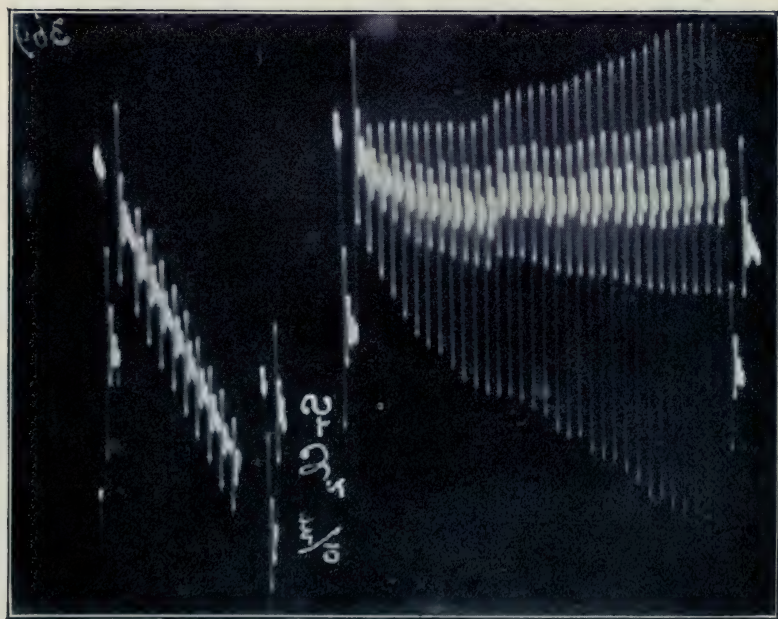
(368) Subsequent effect of Calcium Chloride.



Before. \uparrow After.
 $\text{CaCl}_2 \text{ m}/10.$

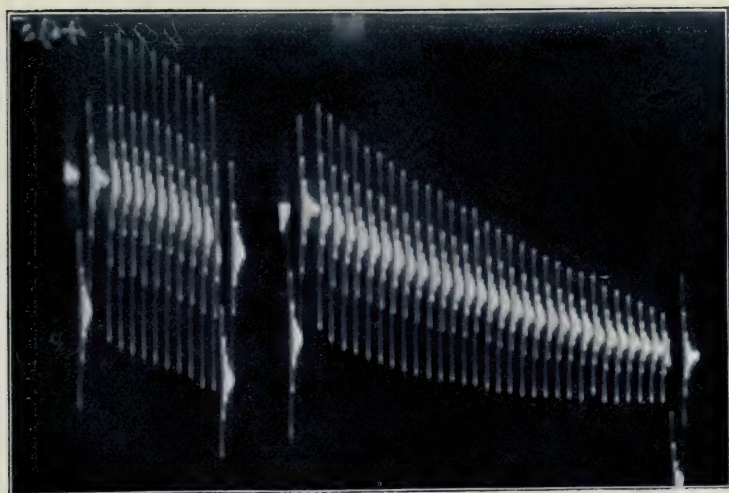
FIG. 26.

(369) Effect of Strontium Chloride.



Before. \uparrow After.
 $\text{SrCl}_2 \text{ m}/10.$

FIG. 27.
(495) Effect of Strontium Bromide.

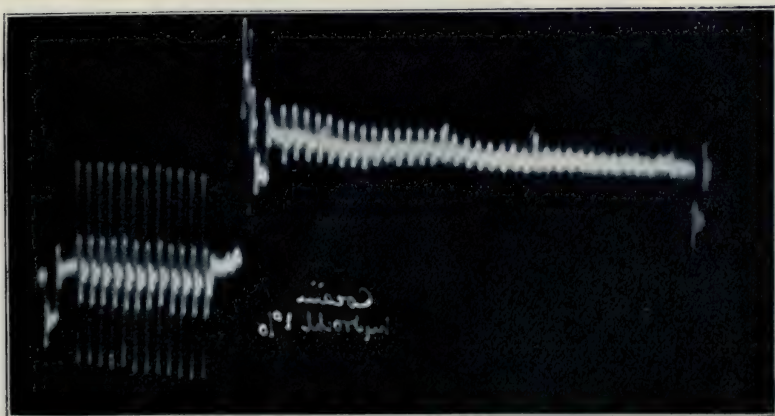


Before.

$$\text{SrBr}_2 \uparrow \text{ni/10.}$$

After.

FIG. 28.
(840) Effect of Cocaine.



Before.

Coc. hydrochl. 1 %.

After.

Before. ↑ After.
Ext. Opii. 5 %.

<i>Ammonium.</i>				<i>Sodium.</i>			<i>Potassium.</i>		
	Am Cl	Am Br	Am I	Na Cl	Na Br	Na I	K Cl	K Br	K I
Norm.	100	100	100	100	100	100	100	100	100
5 min.	76	75	75	68	91	95	50	55	32
10 „	65	65	68	55	82	95	12	22	0
15 „	53	60	61	50	75	90	0	0	0
20 „	41	50	48	37	75	90	0	0	0
25 „	30	—	—	27	—	—	0	0	0

For the table we have—

With K the order I Cl Br.
 With Na „ Cl Br I.
 With Am „ Cl I Br.

This last trio is, I think, to be dismissed; the differences are within the limits of experimental variations and errors of measurement.

The second trio must also be dismissed; the marked effect of NaCl is probably peculiar to the method; a decimolecular solution of NaCl, viz., physiological saline 0·58 per cent., is the starting point of all the nerves, and a nerve transferred from this solution into a solution of 1·16 per cent., so as to be acted upon by a supplementary mass of 0·58 per cent. NaCl, is, so to speak, already inclined in the direction of a NaCl modification; it may be that it is more inclined to an AmCl than to an AmBr modification. I have not tested this second possibility; with regard to the first, however, I am able to say that a second bath produces more effect than a first bath; that a $\frac{M}{5}$ solution produces more than twice the effect of a $\frac{M}{10}$ solution; and the latter more than twice the effect of a $\frac{M}{10}$ solution. Taking number of molecules along an abscissa, and amount of alteration of nerve along the ordinates, the curve of modification, as it first rises, is convex to the abscissa.

The only point at all clear as regards the acid element, is in the first trio, where, according to my experience, KI manifests itself, as a rule, more depressant than KBr. The reverse difference between NaI and NaBr is one that I am not satisfied with, simply because experiments go quickly with K, slowly with Na, so that a clear difference is less difficult to detect in the former than in the latter case.

But for the question of difference between the I, Br, and Cl factors, finer determinations will be required.

As might be expected—as is indeed well known in the case of other tissues—caustic potash, molecule for molecule, is more effective than caustic soda. This will be sufficiently substantiated by the accompanying table, and by a couple of illustrative records:—

790. $\text{NaOH } \frac{\text{M}}{20}$.

720. $\text{KOH } \frac{\text{M}}{40}$.

K OH		(717)	(719)		(720)	(721)
		$\frac{\text{m}}{10}$	$\frac{\text{m}}{20}$	$\frac{\text{m}}{30}$	$\frac{\text{m}}{40}$	$\frac{\text{m}}{50}$
N.	..	100	..	100	..	100
5 min.	..	0	..	0	..	120
10 „	..	0	..	0	..	30
15 „	..	0	..	0	..	0
20 „	..	0	..	0	..	0
25 „	..	0	..	0	..	0
30 „	..	0	..	0	..	0

Na OH		(791)	(790)	(792)		(795)
N.	..	100	..	100	..	100
5 min.	..	0	..	113	..	94
10 „	..	0	..	113	..	81
15 „	..	0	..	107	..	63
20 „	..	0	..	93	..	50
25 „	..	0	..	83	..	38
30 „	..	0	..	73	..	30

It was accidentally omitted in this series of experiments to test $\text{Na OH } \frac{\text{m}}{40}$ and $\text{K OH } \frac{\text{m}}{30}$. But as the two strengths are as 3·3 Na OH to 2·5 K OH, a comparison is possible, bringing out under conditions against the conclusion that K OH is more effective than Na OH.

In repeating series of this kind, it would be better to take a centimolecular unit, and to observe the effects of the scale $\frac{\text{m}}{100} \times 1, \times 2, \times 3, \times 4, \times 5$.

Admitting that the basic element is the predominant factor in a salt, the further investigation of the bromides loses much of its special interest. In relation to isolated nerve, potassium is sedative, whether in bromide, chloride, or iodide; and not a bromide sedative, whether of sodium, potassium, or ammonium, or of all three.

Such, however, is the glamour of the word bromide that I could not omit to test some other bromides, viz., calcium, lithium, strontium, and gold. According to my observations, the first two keep company with sodium, and give very little sedative effect; strontium bromide is about on a par with ammonium bromide; and gold bromide, molecule for molecule, is at least five times as sedative as potassium bromide. But as regards gold bromide, I think it is to be put out of comparison at once; molecule for molecule, it is not much more "sedative" than gold chloride.

The two bromides of most interest at this stage are those of strontium and of potassium.

If a bromide (or other halide) acts only *qua* potassium or sodium, or calcium, or strontium salt, and if there is a true antagonism between, *e.g.*, potassium and strontium, then the bromide of potassium should be antagonised by the bromide of strontium. To discuss this question it would, properly speaking, be necessary first to consider the action of strontium and of calcium; and the antagonism which Ringer has pointed out on the heart between these bases and potassium salts.

I am, however, unable to consider this question briefly, and must defer it to some future occasion, when I shall hope to go into the general question of antagonism.

For the present I must be content to say that *under certain conditions, but not always*, strontium and calcium chlorides cause an augmentation of the nerve response, and exhibit an apparent antagonism to potassium chloride.

The behaviour of the salts towards each other under certain conditions (which, however, I am at present unable to distinctly specify) is well shown by the following experiments:—

366-7-8. Nerve previously bathed for one minute in $\text{KCl } \frac{\text{M}}{20}$.

Subsequent effect of CaCl_2 $\frac{\text{M}}{10}$.

Subsequent effect of KCl $\frac{\text{M}}{20}$.

Subsequent effect of CaCl_2 $\frac{\text{M}}{10}$.

But I cannot always bring off experiments like this; strontium chloride has generally failed to give any improvement of already "good" nerves; on the other hand, it has had a marked reviving effect upon "poor" nerves, and upon the nerves of moribund frogs or of frogs that had recently died naturally, and upon nerves depressed by previous treatment with weak solutions of potassium salts. The most marked action of strontium is, I think, best characterised by the term "revival"; implying, as a previous condition, a depressed state of nerve.

With regard to the antagonism between strontium and potassium, I have generally found it come off in both directions, viz., revival by strontium after potassium, depression by potassium after strontium; but here again failure has not been unfrequent: strontium may fail to restore a nerve too profoundly depressed by potassium; potassium may fail to affect a nerve too fully under the influence of strontium. Duration of immersion as well as strength of solution influences the result; but I have not yet a sufficient experience of prolonged experiments to be able to say whether in these the antagonism has been false or true—a mere washing out of salt by prolonged immersion, or an actual modification of the nervous matter, according as it is impelled in one or the other direction by potassium and by strontium.

Reserving, then, such uncleared points for a future occasion, when I may be in a better position to discuss the problems of antagonism, we may at present recognise: (1) That there is mutual antagonism (whether true or false) between SrCl_2 and KCl ; and (2) an initial augmentation of the nerve response by SrCl_2 under certain conditions, which augmentation is probably a revival of depressed nerve.

The antagonism between SrCl_2 and KCl has generally been clear. But as regards SrBr_2 *versus* KBr , or CaBr_2 *versus* KBr , or *versus* KCl , antagonism (which, if the base were *solely* efficient, might be anticipated) has not hitherto

presented itself in any one instance of a considerable number of trials.

This has been one of the items leading me to the conclusion that in the action of a salt, AB, B is the predominant but not solely active factor, and that the salt acts as a total product, $A \times B$, not as A only, nor even as B only, nor even as $A + B$.

This formal conclusion is, I think, most clearly illustrated by strontium bromide (and perhaps by calcium bromide, but with the latter salt my experiments have been limited). Although the predominant factor, Sr, is in this case an augmentor, the compound, SrBr_2 , is a depressant.¹

369. Effect of strontium chloride $\frac{M}{10}$.

495. Effect of strontium bromide $\frac{M}{10}$.

In dealing with ALKALOIDS I have not made up their solutions on a molecular scale, but have generally taken as starting point a 1 per cent. solution in physiological saline, subsequently working up or down according to the result. And, with one exception (aconitine), I have at present sought to learn only one point regarding any given alkaloid or other hypnotic, viz., whether it is obviously active or inactive upon the nerve-fibre.

To attempt more than this at this early stage would soon have led into difficulties; even the preliminary point just mentioned has not always come out with clear certainty; the activity of an alkaloid under the chosen conditions of experiment has always been clear enough, but its absolute inactivity may not be predicated from the fact that under the same conditions it has not sensibly modified the nerve. It is evidently, in many cases, a question of degree of modification in relation to strength of solution and time of action; it is equally evident that in this case, as in that of salts, a pronounced effect has been more easily and clearly brought into view than a gradual effect, of which we might feel assured only by more careful testing, and more

¹ It is also illustrated by the trio EtBr, EtCl, EtI, the first of which is most excitant, and the last most depressant. (*Vide* Autumn number of BRAIN, p. 285.)

especially on nerves giving before the action of the drug an absolutely level "normal" series of deflections.

In saying, *e.g.*, that aconitine is active and morphine inactive, we may be quite sure of the first point, but far from sure of the second in its absolute sense. The statement is only comparative, and implies that under similar conditions, viz., immersion for one minute in a 1 per cent. solution, an obvious effect or no obvious effect has been produced. It does not involve the assertion that morphine, tested for longer periods by stronger solutions, will be found totally inactive.

The two drugs just named are those selected for immediate demonstration as likely to afford a clear contrast.

EXPERIMENT III.

MORPHINE AND ACONITINE.

Time.	Deflection.	Time.	Deflection.
0 min.	20	8 min.	18
1 "	25	9 "	9
2 "	30	10 "	4
Bath of Morphine hydrochlorate, 1 % solution for 1 minute.		11 "	2
		12 "	1
		13 "	0
		14 "	0
		15 "	0
4 "	35	[90 "	0]
5 "	35		
Bath of Aconitine hydrochloride, 1 % solution for 1 minute.			
7 "	40 (off scale)		

I was somewhat surprised by the inertness of morphine as compared with aconitine, and am not in a position to offer any explanation. One naturally calls to mind that the former is preeminently a centrally acting, *i.e.*, a comparatively weak and, therefore, special sedative; while the latter is preeminently a peripherally acting, *i.e.*, a comparatively strong and, therefore, general sedative.

And from the experimental records I am about to show, it would appear as if the sedative action of opium were

dependent upon some other of the many constituents of opium in higher degree than upon the isolated morphine salt. 567 is a record of the effect (little or none?) of a five per cent. solution of morphine tartrate. 724 is a record of the obviously more considerable effect of a 5 per cent. (or rather less) fresh watery extract of opium (with NaCl added up to 0.58 per 100).

Aconitine is the one drug in which (thanks to Professor Dunstan, who has kindly furnished me with chemically correct samples of aconitine and of its derivatives) I have had an opportunity of, so to speak, hunting down the ringleader of the molecule. I do not say that this ringleader has been at all perfectly hunted down at present, nor that anything more has been done by this short method than has already been accomplished by prolonged pharmacological experiments, nor, indeed (considering the newness of the method), that I could claim any credit to the results, in the absence of such previous experiments on the usual lines.

Aconitine is held by chemists to be acetyl benzaconate. The experiments I have made go to show that it is the acetyl group upon which the activity of aconitine chiefly depends. Briefly stated, the facts are these: aconitine is very active; benzaconine has very little action; aconine is inert; there is a remarkable similarity between the records of aconitine and of acetic acid.

This result, small though it be, is well calculated to draw us on to further enquiries of the sort with chemically correct alkaloids and alkaloid derivatives. When we have ascertained of a given alkaloid that it is active, the next thing we want to learn is by virtue of what group of atoms it acts; to use another metaphor, if an alkaloid or other compound will bite into living matter, how does it show its teeth, and what are its teeth? We shall no doubt have to proceed as in the simpler case of the haloid salts, where we saw plainly enough that the basic element is the tooth, and that potassium is a sharper tooth than sodium. However, at the present moment I have no data in point. I know that cocaine acts, and am told that it is benzoyl-methyl-ecgonine; but I do not know whether it does so by virtue of the benzoin, or

the methyl, or the ecgonine group; I know that physostigmine and gelsemine act, but have no idea in what part of their molecule their teeth are to be found. I can go no further without the good offices of chemists, who happen to be working at pharmacological chemistry. In this connection I think it to be waste of time and confusion of mind to work with commercial products unless specially prepared for this non-commercial purpose, and I do not intend to further pursue the vein except when I may feel assured that clean molecules are placed in my hands. I will therefore close this communication by the bare exposition of some records of the action of alkaloids and other drugs reputed to be sedative, and bearing out the following series of summary statements:—Cocaine is active (840); morphine is less active than extract of opium (567, 724); physostigmine is active (355); chloral hydrate is active (321); butyl chloral hydrate is active (287); gelsemine is active (308); aconitine is very active (665), probably by virtue of an acetyl group, as indicated by the similarity between its record and that of a deci-normal solution of acetic acid (696); certainly not by virtue of aconine (666), which is inactive; veratrine is very active (3,122), and, to superficial examination, very similar in action to aconitine; curarine is active (3,126).

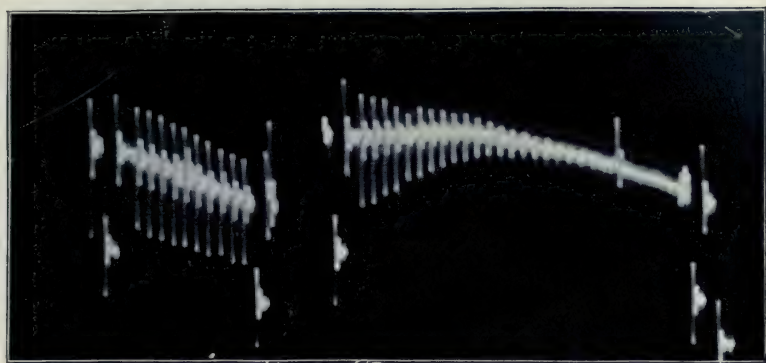
Addendum.

I may take this occasion to print four additional records of observations to which allusion was made in the autumn number of this Journal.

Fig. 40 (Obs. 256) is an instance of "false antagonism," and should, properly speaking, have been given in connection with the remarks made upon p. 287. The first part of the record gives the normal; the second part gives the effect of sodium chloride solution of semi-molecular strength (2·4 per cent.); the third part gives the subsequent effect of distilled water, which, presumably, washes out the excess of salt.

Fig. 41 (Obs. 3,195) gives the effect of a 1 % solution of digitaline in normal saline.

FIG. 31.
(355) Effect of Physostigmine.

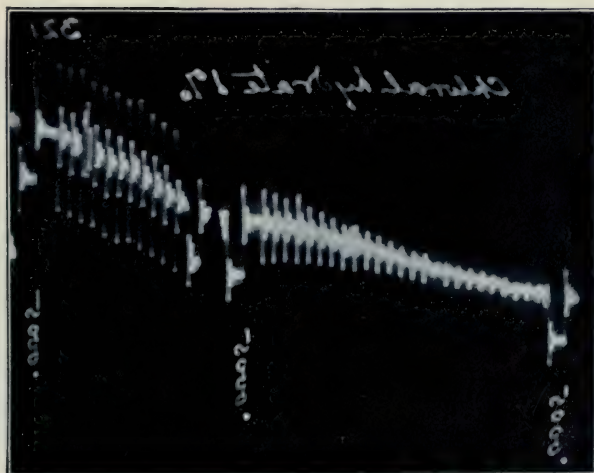


Before.

↑
Physostig. 2 %.

After.

FIG. 32.
(321) Effect of Chloral Hydrate.



Before.

↑
Chlor. Hyd. 1 %.

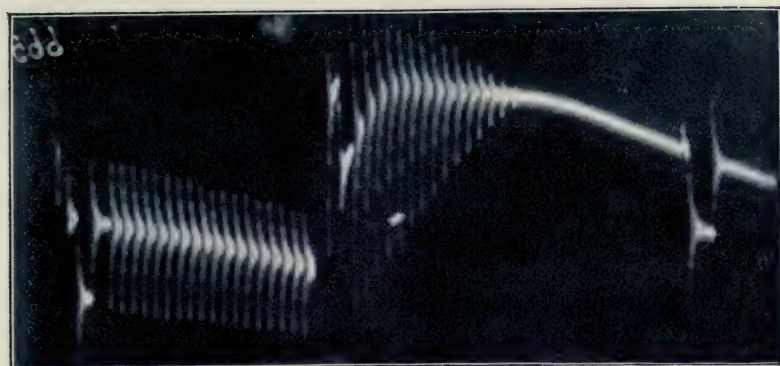
After.

↑

55 minutes
900

↑

FIG. 35.
(665) Effect of Aconitine.

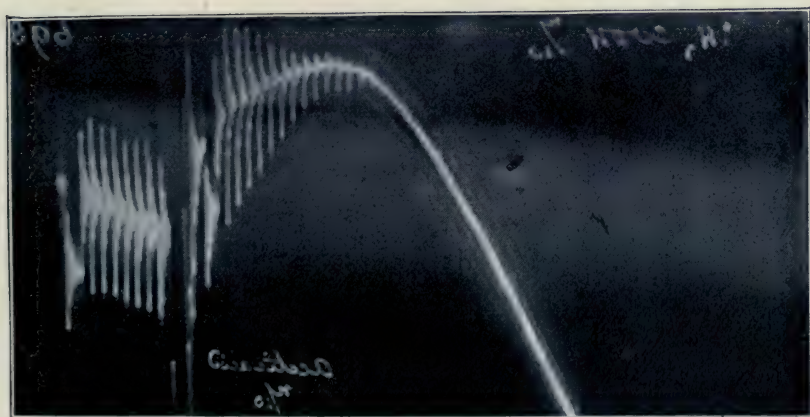


Before.

↑
Aconitine 0.33 %.

After.

FIG. 36.
(696) Effect of Acetic Acid



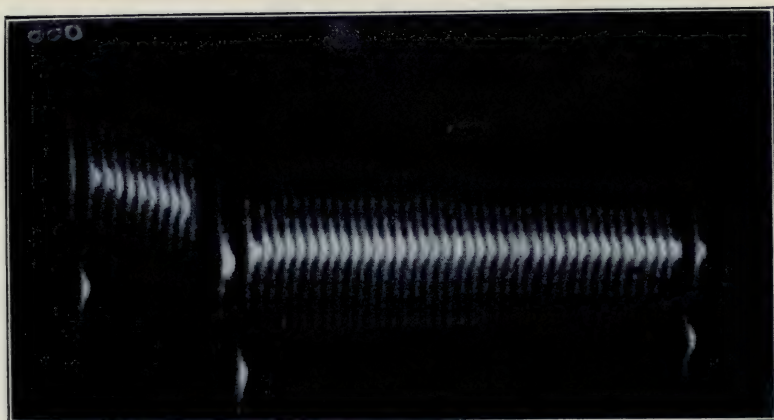
Before.

↑
 $\text{CH}_3\text{COOH. } n/10.$

After.

5149

FIG. 37.
(666) Effect of Aconine.



Before. ↑
Aconine 1 %.

After.

FIG. 38.

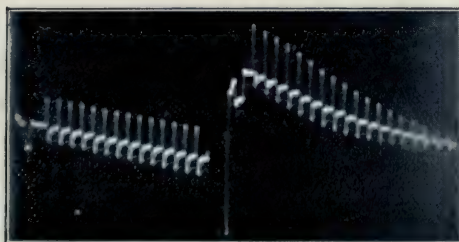
3,122) Effect of Veratrine on a series of polarisation increments.



Before. ↑ After.
Veratr. Hydrochl. 1 %.

FIG. 39.

(3125) Effect of Curare.



Before. ↑ After.
Curarine 1 %.

FIG. 40.
(256) False antagonism between H_2O and NaCl.

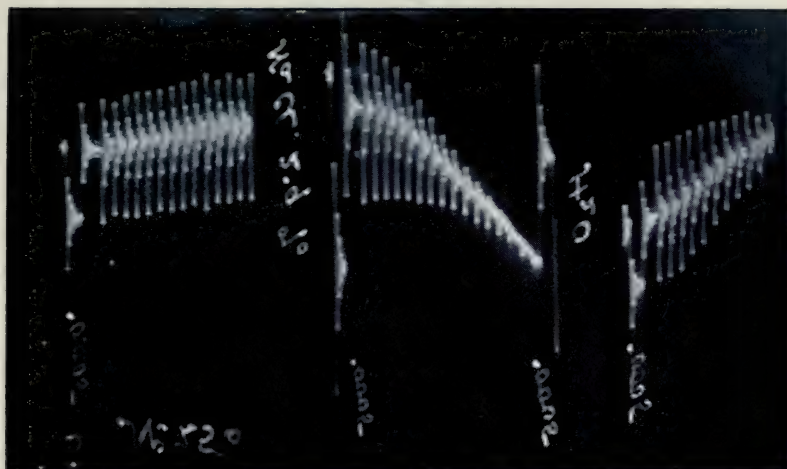
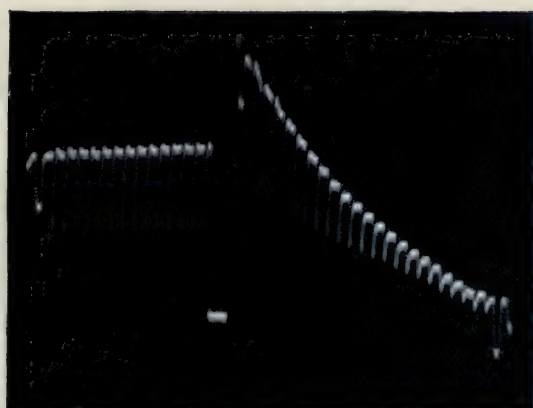


FIG. 41.
(3195) Digitaline 1 %.



Before.

After.

FIG. 42.

(3196) Convallamarine, 1 %.

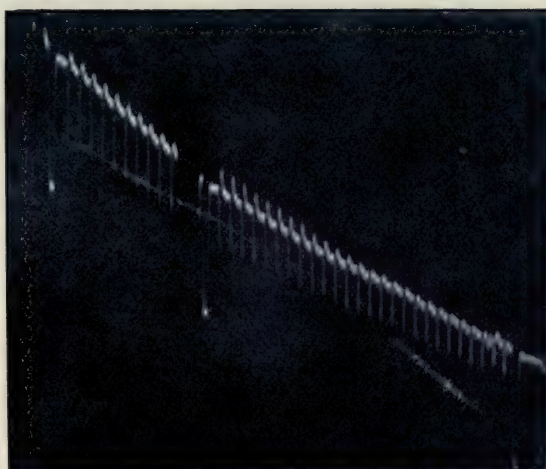


FIG. 43.

(3128) Strychnine sulphate, 1%.

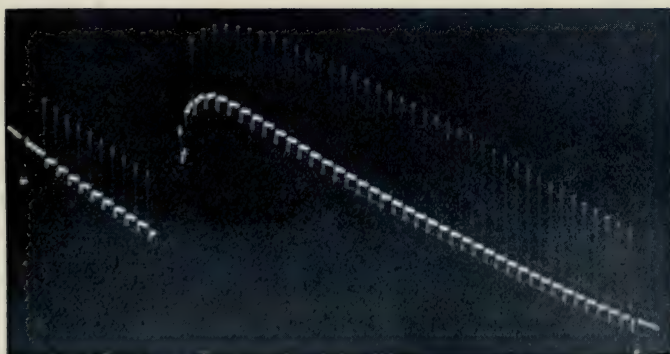


Fig. 42 (Obs. 3198) gives the effect of a 1 % solution of convallamarine.

Fig. 43 (Obs. 3,128) gives the effect of a 1 % solution of strychnine sulphate.

Some apology, or rather justification, seems perhaps called for with regard to the large number of illustrations contained in this paper as compared with the small amount of verbal comment. Such justification consists in the fact that each individual record is, in effect, an autobiographical episode related by the nerve itself, in language clearer, briefer, and more trustworthy than the most exact verbal descriptions and columns of numbers.

FOUR CASES OF CEREBELLAR DISEASE [ONE AUTOPSY] WITH REFERENCE TO CEREBEL- LAR HEREDITARY ATAXIA.

From the Wistar Institute of Anatomy and Biology.

BY WILLIAM G. SPILLER, M.D.

*Neurologist to the New Jersey, and Pathologist to the Pennsylvania, Training
Schools for Feeble-Minded Children; Associate in Clinical Medicine,
William Pepper Clinical Laboratory (Pa. Univ.).*

THESE four cases are derived from the Pennsylvania Training School for Feeble-Minded Children. The first three we have studied with Dr. Charles K. Mills during his term of service at the institution, and we take great pleasure in expressing our gratitude to him for the opportunities so generously offered, and for the privilege of reporting these cases in this paper. We are under no less obligations to Dr. Henry W. Cattell, who, as pathologist of the institution, has been able to place at our disposal the fourth important case, which he has in part already made public.

To all those in authority at the school, especially to doctors Barr, Llewellyn, White, and Morris, we acknowledge our appreciation of cordial assistance, without which the study of these cases would have been impossible.

CASE I.—The first patient attracts attention chiefly from the position which he voluntarily assumes. He moves about the room on "all fours," as shown in the photograph (see fig. 1), and his power of locomotion in this way is about equal to that which a normal individual would manifest in the same position. He is, therefore, in no degree paralysed. The agility displayed in the use of the lower limbs under the conditions mentioned forbids any such supposition. Observe the patient, however, standing erect (see fig. 2), and on a superficial examination the idea of

paralysis, or rather paresis, presents itself to the mind of the examiner. His body inclines backwards, and he seems in danger of falling backwards at any moment; a plumb-line dropped from a point between the shoulders would hang some little distance from the lower extremities. This tendency to fall backwards has been noted in destruction of the cerebellum (Luciani).¹ He stands with his legs wide apart, in order to broaden the base of sustentation, as is seen also in an animal in which the cerebellum has been removed and which at first is completely unable to stand, and learns to do so later by great abduction of the legs (Luciani). He seeks support with his upper extremities and, if permitted, grasps the nearest object, and at a moment when his equilibrium is more perfect he ventures to take a step. He is awkward and stiff to an extreme degree. As soon as his foot has been placed again upon the ground, with some swaying of the body he regains his balance and, after a certain amount of hesitation, he makes a further attempt at walking. Even in this manner he can only take a few steps. Compare, however, this extreme want of coördination with the freedom of movement in the position of "all fours," when the centre of gravity is lowered and the base of support is enlarged. In walking in the erect position, which he never attempts voluntarily, the lower limbs are stiffly held, and this stiffness persists only during the movements of walking. The gait is of the pelvic type, as in the cases of Klippel and Durante,² *i.e.*, the movement is chiefly from the hip-joints. The sway of the body is not increased by closure of the eyes. Romberg's sign is therefore absent. When he lies flat upon his back, the movements of the extremities are more free and the want of coördination is less perceptible. The incoördination may also be observed in the movements of the upper extremities, but a point worthy of note is the less degree of this in comparison with the incoördination of the lower limbs. He can carry a glass of water to his mouth, but spills the contents more or less, and is able to pick up a small object.

There is a tremor during repose and in movement, and this is not increased in attempting to grasp an object. He can use a spoon, but is not trusted with knife and fork, and cannot handle tools. It is probable that the false nystagmus, the "saccaded movements" of the eyeballs which occur whenever he attempts to look attentively, interfere greatly with coördination. This myoseismia is not seen in the movements of the muscles of vegetal function. The term "fibrillary chorea," employed by Klippel and Durante, describes his tremor quite well.

The musculature of the body, as may be observed from the photograph, is well developed, especially in the upper half, and there is no atrophy anywhere, no muscular rigidity, and no contracture when the limbs are at rest. The lower extremities are small in proportion to the rest of the body.

The knee-jerk is greatly exaggerated, and ankle-clonus is present on both sides. In the upper extremities the reflexes are likewise increased. The abdominal and plantar reflexes are present. We could obtain no cremaster reflex, but this may have been the result of unfavourable conditions. The sensation of pain and touch is normal everywhere. Vision is defective; he is not able to regard an object steadily, and cannot recognise colour, although he can recognise form. He has never learned the alphabet and therefore cannot read. Strabismus is not present. In looking to one side he has marked nystagmus, but of a peculiar type. It is present also in regarding an object attentively directly in front of him, and is horizontal as well as vertical. It is an incoördination of the eyeballs and is similar to the incoördinate movements of the limbs. It is like the nystagmus in the three cases reported by Klippel and Durante (*loc. cit.*), which they describe as false nystagmus, or as a series of "movements saccadés," and not rhythmical. There is no Argyll-Robertson pupil, and the pupils respond promptly to light, and probably to accommodation, though it is difficult to get the patient to fix an object.

The report from Dr. Risley states there is a "constant nystagmus which does not follow the rules of ordinary nystagmus and is more violent on every attempt to fix an object, and ends with a general rotary movement of both eyes. It is simply a want of coördination. The right disc is paler than the left, and the vessels are smaller. There is no strabismus." The hearing is very good.

The speech is very peculiar; he talks freely, but the articulation is very imperfect and to a stranger incomprehensible. It can hardly be called jerky and it is not explosive, as in the next case we report. He does not hesitate and then begin again by repeating the last word, and the comparison with the speech of one who is in great cold (Klippel and Durante) is hardly applicable here. He does not omit syllables, and repetition is possible but indistinct.

His general condition appears to be excellent. The vascular system is in normal condition. There are no trophic disturbances, and no subjective sensations of pain.

The patient's intelligence is of a very low grade. This feebleness of intellect is noted especially in two, although present in all four cases we report. He understands only very simple questions, and replies correctly. His expression is one of stupidity. His habits and character are commendable. It is, perhaps, worthy of note that while nothing very characteristic may be noticed in the shape of his head, the occiput is very prominent. The hard palate is exceedingly arched. We learn from the record given by those who previously had charge of him that he was born at full term in a normal, non-instrumental labour, that he was the fifth child, and that as a baby he was considered healthy. The supposed cause of his condition was a fall at the age of nine months. We further find that he did not have any of the infantile diseases; an important fact in view of what we have to say later. He had epileptiform convulsions at the age of three years, but these have apparently ceased. He did not begin to walk until he was five years old. It was not until he was fifteen or sixteen that any peculiarity was noticed, when lack of power of locomotion and a rolling upwards and sideways of the eyeballs attracted attention. Probably, however, certain earlier signs had been disregarded by his relatives. When we learn that one child in this family died at birth, another in the second month from pneumonia, a third in the seventh month from marasmus, a fourth on the third day, and finally that one child died in the eighteenth month from "defective circulation," we are certainly justified in regarding this patient's condition as congenital. The father was twenty-eight, the mother twenty-four at the birth of this child. There is no history of alcoholism, insanity or imbecility in the family. The mother died of abscess of the liver; the father is still living. This case does not support the opinion held by some that great difference in the ages of the parents may be an accessory cause of imperfect formation of the central nervous system. The patient's age at present is twenty-three.

CASE II.—W. C. T. attracts attention by his gait and speech; and the possibility of multiple sclerosis beginning in childhood, as described by Unger, Mendel, Marie and Oppenheim, has been present in our thoughts. There is no nystagmus, either real or false, such as in the previous case, in which the movements are more a series of interrupted jerks of the eyeballs. The patient is able to regard an object steadily; he recognizes colour and form, he knows the alphabet, but cannot read, although he can make a few letters. Sight is apparently very good, and there is no diplopia and no paralysis of the external eye-muscles. Hearing

is also good. The eye report from Dr. Risley is as follows: "Left eye: Entire eye-ground is filled with glittering striæ giving shot-silk appearance. Arteries and veins are both small. The nerve, particularly the outer half, is grey; lamina cribosa grey and devoid of capillarity. Inner half of nerve is slightly pinkish, but its inner margin is obscure. Eye has low grade of hypermetropic astigmatism. Right eye: Retina does not present shot-silk appearance, but is flannel-red and fluffy. Veins are rather full, and arteries actually too small. Outer margin of nerve is grey, but the inner half is bright red, and fades almost insensibly into the surrounding eye-ground. The case probably is one of retro-bulbar descending neuritis, now in its retrograde stage, and the process is further advanced in the left eye.

The speech is most peculiar; it is scanning, explosive and indistinct; some words seem to come as if forced out by an increased effort. The knee-jerk is much exaggerated on both sides; the superficial reflexes are preserved. There is great incoördination in all the limbs on motion. The gait is decidedly cerebellar in type, and he staggers as a drunken man; this is especially noticeable in turning round. In the movements of the upper extremities there is a tremor not noticed when the patient is at rest; but this does not prevent the use of knife and fork, although he is not skilful in the use of tools.

This can hardly be called a true intention tremor, although it does occur only when the arm is in motion; but it does not increase as the object desired is approached. It begins at once on movement of the limb. It is an expression of incoördination.

In standing erect the station is not affected by closure of the eyes (Romberg's sign). The patient is not weak, the hand-grip is strong, and resistance to passive movements in the lower extremities is considerable. It is not improbable that he has vertigo, but his statements are not convincing. He occasionally soils and wets his clothing, which may possibly indicate some weakness of the sphincters; more probably, however, this is due to want of attention. An important statement which he makes, in whatever way the question may be put, is that he occasionally falls down, and always on the left side, with an inclination forwards or backwards. It has been noticed in operations on the cerebellum and in cerebellar disease, that there is frequently a tendency to fall towards the affected side; the rule, however, is not invariable. An animal in which one-half of the cerebellum has been removed is so weak on the operated side that it appears hemiplegic (Luciani). In Lloyd's³ case of cerebellar tumour

recently reported the patient, whom we had the honour of seeing, fell towards the side on which the tumour was. Sensation for pain and touch is normal.

The history, as given, informs us that this patient was born at full term in very difficult labour. There was no history of alcoholism, insanity or imbecility in the family. There is no great difference in the ages of the parents. The patient did not begin to walk until the age of 7, and now can go up and down stairs with the aid of the banister. He has never had eleptiform convulsions, but has had pneumonia three times. This we mention as patients with chronic cerebellar disease seem frequently to be liable to some pulmonary affection. His intelligence is only of medium grade. He is now 13 years old. While we have no history of family disease, the fact that he was not able to walk until 7 years of age indicates that his condition was either congenital or began soon after birth.

The condition of the reflexes excludes the probability of Friedreich's disease, but the possibility of an early development of multiple sclerosis must remain, although the diagnosis of cerebellar disease seems more probable. The first attempts to walk at the age of 7, the speech more explosive than scanning, the gait so distinctly cerebellar in type, possibly the tendency to fall towards one side, are facts in favour of some disease of the cerebellum, either congenital or dating from early infancy. However, the case reported by Royet and Collet,⁴ in which the diagnosis of disseminated sclerosis was given, and which proved at the autopsy to be a case of cerebellar disease with no apparent morbid changes in the cord (microscopic examination was not made), must render one cautious in distinguishing between these two forms; and as Brissaud⁵ says in regard to such a combination of symptoms as seen in the above case, when heredity fails the diagnosis must be very doubtful.

CASE III.—The patient, W. C. S., is a girl of 7 years. The circumference of the head through the glabella and lambda measures seventeen and a quarter inches. She has very feeble intellectual power, is not able to speak, and seldom gives utterance to any sound. She is said to come when she is called; the sense of hearing is probably, therefore, not absent. She was born in a syncopal state at full term, but in difficult labour. Convulsions began a year after birth. She began to fail in health when four months old. There are no other children in the family. A brother of the father is feeble-minded. When nearly

4 years of age she had a severe attack of epileptic convulsions, and lay in a condition of status epilepticus for several hours. The history states in a very indefinite way that she had had "brain-disease." The musculature is of very slight development. Sensation for pain is preserved. The pupils react to light, and probably to accommodation. There is no true nystagmus, but rather what might be described as instability of the eyeballs. These turn in every direction, and when she looks to one or the other side there is slight nystagmus-like movement. Her vision is apparently good, as she can see to pick up large objects. There is no paralysis of the external eye-muscles. Dr. Risley made various unsuccessful attempts to see the eye-grounds. He succeeded in obtaining a glimpse of the nerve of the right eye, and thought it was normal in colour.

She stands with her legs far apart, and with knees slightly flexed; her station is very insecure, and there is a constant tendency to fall. The gait is not spastic. When she first stands up she is obliged to take a few short steps in order to gain her equilibrium, but when sitting on the floor she can move about with great agility and considerable speed. She has full power of motion in all her limbs, but the movement is very inco-ordinate. In standing or walking she inclines forwards, and is very apt to fall when she approaches an object. The instability of her eyeballs accounts for some of the incoördination of movement. This tendency to fall forwards has been noted in other cases of cerebellar disease. It was present in Sommer's⁶ patient. She did not begin to walk until she was nearly four years old. She can pick up large objects with either hand with great difficulty, and constantly lets them fall. The knee-jerk is exaggerated on both sides, but there is no ankle-clonus. She is a demi-microcephalic, with symptoms of involvement of the cerebellum. How much of this condition is due to the brutal treatment the mother received during gestation is, of course, unknown.

While the three cases we have just described seem to be the manifestations of cerebellar disease, we cannot overlook the important fact that disturbance of co-ordination has been observed in cerebral diplegia, as Higier,⁷ though not the first, has recently so clearly pointed out. He has also collected a number of cases in evidence of this fact. These cases of ours resemble in many ways those of Nonne.⁸

Redlich⁹ has described a case of cerebellar atrophy which



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

in many respects is not unlike these cases we report, and which is of special interest to us, as we have seen and studied his patient. In the history given by him there is mention of some form of brain disease at the age of two, and the child was obliged to walk on "all fours" until he was twelve years old. In none of our cases can we consider the diagnosis of Friedreich's disease probable, and yet we must remember and agree with the opinion of Schultze,¹⁰ that the case reported by Menzel¹¹ may be regarded as a connecting link between the cerebellar and spinal forms of hereditary ataxia, and that it shows that the two diseases are not always clearly separable.

It seems that these three cases which we report represent some form of cerebellar disease, if not congenital, at least beginning early in infancy; and in all probability of a hypoplastic or sclerotic nature, or both. It is exceedingly probable that the cerebrum is also involved in the process, at least in two of the cases, on account of the very feeble intellectual qualities.

CASE IV.—The fourth case is one of great interest, as an autopsy was obtained. The brain was presented by Dr. Henry W. Cattell as a gross specimen before the Pan-American Congress, and the report was published in the *Transactions* of this body in vol. 1, 1893, p. 365, with the photograph which we reprint. Dr. Cattell reports: There is almost entire absence of the cerebellum and great deficiency of the corpus callosum, in a male of 19 years, who presented incoördination of movements and mental disturbances.

L. H., male (see fig. 3) was the fourth child of healthy native-born parents. No instruments were used at his delivery, which was at full term. As a babe he had a weak back, and did not learn to walk until he was three years old. When five years old he had an attack of scarlet fever and diphtheria, which interfered with his speech and walking, and are supposed by his parents to have been the cause of the child's trouble, though his not walking until three years of age would lead one to think otherwise. He never had any convulsions, but often complained of dizziness. His gait is described as showing a slight stagger, as though he were not sure of his steps, the feet being raised very high from the ground. His habits were good, and he had little to say except

when spoken to. He was able to use his hands. His school progress was interfered with by defective eye-sight, the nature of which was not determined, though he had internal strabismus. The reflexes were normal. L. H. was a member of Company C, which is made up of middle and low-grade boys, varying in age from twelve to twenty. Regular drills of an hour each are held daily. Capt. Weaver, the commander of the battalion, says that his gait was a very peculiar one, in that his steps were short and quick, his trunk stiff and slightly bent forwards, his head fixed and turned slightly towards one side. He had little or no idea of time, but was able to make easy movements on the march and in the drill, though never with any great degree of accuracy. He finally succumbed to tuberculosis.

At the *post-mortem* (Cattell, No. 555; Elwyn, 126) the body was found to be very emaciated; head that of a microcephalic, with cerebellar fossæ quite deep. Circumference of head, $18\frac{1}{2}$ inches; nasion toinion, $11\frac{1}{4}$ inches. The cavity for the cerebellum measured $2\frac{7}{16}$ inches across. From the centre of the middle clinoid process to the internal occipital protuberance measured $2\frac{1}{2}$ inches. Weight of brain, $32\frac{1}{4}$ ounces. In removing the brain one was at once struck with (1) the great deficiency in the corpus callosum, the brain hardly being held together; (2) the small size of the cerebellum, the two lobes being practically of the same size; (3) the atrophy of the left cerebral hemisphere (see fig. 4).

There have been cases recorded in which the cerebellum was very small. A remarkable case was reported by Com-bette¹² in which it was absent, and its place was occupied by a cyst; yet this patient lived to the age of 11 and was well developed. Ferrier¹³ also reports a case in which the left lobe of the cerebellum was a mere papilla, the vermiform process was a minute nodule, and the right lobe was only half a square inch in superficial area and a quarter of an inch thick at the base. The width of the cerebellum from one side to the other, as measured by us from his life-size picture, was $2\frac{1}{16}$ inches, while the width in case we report in a section representing the greatest development, is $1\frac{11}{16}$ inches.

The area just below the bulbo-pontal junction to either side of the anterior pyramids, so prominent in most brains, in this case was depressed, as the lower olives were not developed.

The pyramidal tracts were covered by so few transverse pontal fibres that they could be traced without difficulty into the cerebral peduncles as two prominent bands of fibres. The cerebellum was equally diminished in all parts, and the surface was unusually even. As it had been kept a long time in alcohol we were not able to judge of its original firmness, but the sclerotic area in each hemisphere, and in the vermis must have produced a sensation of greater density. The left posterior corpus quadrigeminum was much smaller antero-posteriorly, and slightly so transversely than the right corpus. This may be of importance, for we shall see that the left anterior cerebellar peduncle and left lateral fillet are also smaller than the right. In Moeli's¹⁴ case the lower corpus quadrigeminum opposite the side of the hypoplastic cerebellar hemisphere and the corresponding lower fillet were also smaller. The oblongata in our case was considerably below the normal size. The cord would hardly be considered very diminutive, although below the normal. The cerebral peduncles were small, and the cerebral hemispheres, especially the left, were not of the size one would expect in a person of 19. The entire cerebral stem was cut in serried sections, from the medulla oblongata to the corpus callosum.

The stains employed were hæmatoxylin, as used by Weigert and Pal, and the method of Rosin was found to give better results than carmine, as the brain had remained so long in alcohol.

Microscopically, the sclerotic area in each cerebellar hemisphere occupies the median external portion; on the left side, where it is more extensive, it involves the posterior part of the dentatum; on the right side the dentatum is not damaged, as the sclerosis is posterior to this body. The different layers of the hemisphere are equally diminished. We have not been able to notice the great variation in the size of the different layers mentioned by Russell¹⁵ in the report of the puppy's cerebellum which he examined. In an atrophied cerebellum from a cat studied by him this great difference did not exist. In Moeli's¹⁶ case, also, the cortex of the hypoplastic cerebellar hemisphere was not

normal in its layers. The molecular and granular layers in our preparations, as in a normal cerebellum, are best developed as the summit of the lobules, and least so where they form the bottom of a fissure. The cells of Perkinje are numerous and well formed; but they are not as near together as under normal circumstances. In the sclerotic areas none of the layers of the cortex are present; in place of these, fine fibres stained black by the hæmatoxylin are observed tangled together and without a regular course.

Coarser fibres are almost entirely absent in these areas. Cramer¹⁷ reports the same condition. No cells of Purkinje may be seen here. In one place an irregular line of what appears to be foreign matter, under low power, is seen to consist of medullated, broken fibres, coarser than the rest when viewed with a more powerful lens. Bands of wavy fibrillæ are found, stained red by the Rosen method, which are not unlike some of the wavy bands of fibrils seen in the cord when coloured by Weigert's new neuroglial stain. Some of these are the sheaths of thickened vessels, others neuroglial fibres. The vessels in these sclerotic areas are more numerous, as they are also in a few other places in the oblongata. This has been reported by Arndt,¹⁸ and he believes that these cases of cerebellar sclerosis are probably of vascular origin. Nutrition is disturbed, the nervous tissue is destroyed, and the neuroglia proliferates. It is however, a known fact that sclerosis of small vessels may be secondary to sclerosis of the surrounding tissue; and Redlich¹⁹ has recently emphasized the improbability of the primary nature of the sclerosis of the vessels in multiple sclerosis. Oppenheim,²⁰ in speaking of syphilis of the central nervous system, says that the supposition has been expressed that the induration which is observed in syphilis occurs when the blood-supply of the nervous parenchyma is not entirely cut off, but only interfered with, and when the process is a gradual one, but that this is doubtful. The vessels have been found diseased in a number of cases of cerebellar sclerosis (Schultze, Arndt, Cramer, &c.). In our case there is no thickening of the small vessels external to the sclerotic areas.

As only the posterior part of the left dentatum is involved in the sclerosis, the remainder of it and the entire right corpus are normal, and contain many nerve-cells. The dentata seem to be resistant to destructive processes; in Arndt's case (*loc. cit.*) they were normal in the midst of greatly degenerated tissue; they were also preserved in the advanced cases of Pierret and Duguet (quoted by Arndt). They were not perfectly intact in Menzel's case. The condition of these bodies explains the absence of degeneration in the right anterior cerebellar peduncle and the partial destruction in the left, although, according to Luciani,²¹ some of the fibres of these bundles come from the middle and lateral lobes of the cerebellum. Ferrier and Turner²² believe that the majority of these fibres come from the dentata. Ferrier²³ makes the statement that the quadrigemina are supposed to be connected with the cerebellum by means of the superior cerebellar peduncles. A connection with the pons, from the results of investigators, seem to be by means of the lateral fillet; this was found degenerated also in some of the recent experiments on the cerebellum made by Thomas.²⁴ The left posterior corpus quadrigeminum was distinctly smaller, and we have found the left lateral fillet smaller than the right. Bechterew²⁵ believes that the posterior corpora quadrigemina have some importance in the coördination of movement.

The right nucleus ruber appears in some sections slightly smaller than the left, but the difference is very little, and in other sections not noticeable. Even in normal brains a slight difference may often be observed in these nuclei, possibly from a slight difference in the level of the section on the two sides.

The fibres within the two nuclei seem to be equal and not fewer than in normal brains. As the two sides of the cerebellum were equally diminished there is no possibility of comparing an atrophied with a sound side, and all parts of the central nervous system in this case, including the cerebrum, are small.

The left thalamus is considerably smaller than the right. Mendel²⁶ has shown that the opposite brachium conjunctivum

degenerates from a lesion in the pulvinar. Ferrier and Turner (*loc. cit.*), from their experiments, believe that the brachium conjunctivum passes from the cerebellum to the nucleus rubra and pulvinar of the opposite side, and that decussation is complete in the nucleus rubra. Thomas,²⁷ also, has recently traced these fibres into the thalamus. Luciani (*loc. cit.*) is not inclined to believe that the fibres to the thalamus are so very numerous. According to Professor and Madame Dejerine²⁸ the thalamus is also connected through the anterior cerebellar peduncle with the same side of the cerebellum, by means of an uncrossed band of fibres. This at first thought seems to explain the diminution in the left thalamus, as the left anterior cerebellar peduncle was partly atrophied; but the difference in the two thalami is proportionally much greater than that in the two peduncles, and we cannot believe the explanation is to be found in this way. The diminution of the left pulvinar is probably due to the diminution of the entire left hemisphere, for the thalamus is certainly closely connected with the cortex. The lenticular nuclei are of the same size on the two sides.

The preservation of the dentata, except in the posterior part of the left corpus, in our case has importance in regard to the atrophied condition of the lower olives. Russell²⁹ mentions in his paper that no changes were found in the lower olives, notwithstanding the alterations of the cerebellar cortex. He regards the integrity of the olives as due to the integrity of the dentata, and states that "the intimate relationship which exists between the corpus dentatum and inferior olive has been too abundantly proved by experimental and other evidence to need further emphasis." In a defectively developed cat's cerebellum he found the dentatum greatly affected, and the opposite lower olive was extremely diminutive. Both the lower olives are so small in our case that, instead of a prominence on the external surface of the oblongata, a considerable depression was observed, and ganglion-cells are only found in the posterior part of the posterior fold. The anterior fold consists of a sclerotic mass without ganglion-cells. A limited number

of cerebello-olivary fibres are only found entering the posterior part of the posterior fold. The fibres of this tract (Schlesinger³⁰) are to be regarded as connected chiefly with the ganglion cells of the ventral fold of the olive. These fibres are in large part deficient. Fibres from the lower olives pass chiefly to the dentata, but it is possible that a portion of these pass directly to the cerebellar cortex (Bechterew, *loc. cit.*, p. 130).

Since Gudden has shown that atrophy of the opposite olive follows lesions of the cerebellar hemisphere, this atrophic condition has been frequently observed. The vermis is very small and sclerosed, and the tegmental nuclei cannot be seen.

The posterior and anterior accessory olives are in a nearly normal condition. In Arndt's case (*loc. cit.*) the ganglion cells were almost entirely absent in the inner accessory olive. Ferrier and Turner (*loc. cit.*) found the accessory olivary nuclei atrophic, after extirpation of a lateral cerebellar lobe. Few fibres are found in the raphe between the lower olives, as Schultze³¹ also mentions in his case. While the lower olive opposite the hypoplastic cerebellar hemisphere was greatly diminished in Moeli's³² case, the inner accessory olive was only moderately diminished and the posterior accessory olive very slightly so.

The clavate and cuneate nuclei contain many cells. It is difficult to say if they are present in normal number. Amaldi³³ has recently found deficiency of these nuclei on the side of the cerebellar lesion, especially noticeable in the nucleus of Burdach. In Cramer's case (*loc. cit.*) of unilateral cerebellar atrophy the clavate and cuneate nuclei of both sides contained atrophied cells, which were more numerous on the side opposite the lesion. He also found in Moeli's case atrophy of both nuclei of the posterior columns, on the side opposite the lesion.

The pia of the cerebellum, pons and oblongata is not thickened, and its vessels can hardly be said to be sclerotic, although many of them, as well as those within the nervous tissue, are distended with blood.

The lateral nuclei of the oblongata contain numerous

cells; these were few in number in Cramer's case. Ferrier and Turner (*loc. cit.*), on the other hand, found the cells of the lateral nuclei intact after removal of the cerebellum.

The arcuate nuclei are entirely absent, and there are no external arcuate fibres. There were only a few fibres in Arndt's³⁴ case; the arcuate nuclei, also, were almost entirely without cells, and the striæ acusticæ were only faintly developed. We can find no fibres of this latter band. The nuclei of the posterior columns were perfectly intact in Arndt's case: he is, therefore, embarrassed in attempting to give an explanation of the almost complete atrophy of the external arciform fibres. Cramer reports atrophy of these fibres on the side of the cerebellar lesion. Arndt suggests that descending fibres may be contained in these tracts. The atrophy of the lateral and arciform nuclei he cannot explain. If we accept the theory of Menzel³⁵ and Arndt, that external arciform fibres arise in the cerebellum and terminate in these nuclei, we must assume that the degeneration of the cells of these nuclei is tertiary. The possibility of tertiary degeneration is held by some histologists, especially for the motor cells of the anterior horns of the cord in cases of old hemiplegia.

The striæ acusticæ are frequently absent under normal conditions. Bechterew³⁶ believes that these striæ enter the arciform nuclei and form part of the arcuate fibres of the opposite side. They are in close connection with the cerebellum, and not with the acoustic nerve, however. Koelliker³⁷ says it is certain that the greater portion of these striæ are in connection with the cochlear nerve and unite it with the cerebellum, and that v. Bechterew is mistaken when he denies this. Ramón y Cajal³⁸ is fully in accord with the statement of Held and Koelliker in regard to the existence of axis cylinders in the striæ acusticæ from certain of the cells of the tuberculum acusticum, and in his recent work gives an excellent picture of these fibres. In Menzel's case of cerebral atrophy these striæ were imperfectly developed. The acoustic nerves, both branches, and the ventral nuclei, in our case, appear normal. The statements concerning

the condition of these formations, in the reported cases of cerebellar sclerosis, have not agreed; but Arndt regards the existence of a connection between the acoustic nerve, and especially the medial branch, with the cerebellum by means of the direct sensory cerebellar tract as certain.

The superior olives offer a decided contrast with the lower olives. The sixth centres and the fibres connecting these with the superior olives appear to be normal. Bechterew (*loc. cit.*, p. 58) regards the superior olives as centres of control for the reflex movements of the eyeballs. In favour of this view, in his opinion, is the connection of the superior olive with the cerebellum, for more or less extensive lesion of the cerebellum causes disturbance of the position and of the reflex movements of the eyeballs.

The patient whose nervous system we are studying, had left internal strabismus from paralysis apparently of the left external rectus muscle, but we have not been able to satisfy ourselves that either one of the sixth nuclei or nerves is atrophied; the superior olives, as stated, are intact. Strabismus and nystagmus, according to Luciani, are signs of irritation in cerebellar disease. Although degeneration of the sixth nerve within the pons cannot be clearly demonstrated, we have not been able to examine the extra-bulbar portion. It is not impossible, although we have no proof, that a condition similar to that described by Dejerine and Petrean³⁹ may have been present. These authors found great alteration of certain cranial nerves at and beyond the point of emergence from the oblongata and pons, although the intra-medullary portions appeared intact.

On account of the intense atrophy of both lower olives we have examined carefully the central tegmental tracts. Bechterew⁴⁰ described these bands of fibres in 1885. Arndt also reported atrophy of these tracts in his case. Cramer could not notice any difference on the two sides of the pons, nor could Menzel, although the olives were much atrophied. Mills presented specimens at the Philadelphia Neurological Society (April 23, 1894) of descending degenera-

tion of one of these tracts, from a lesion in the thalamus and internal capsule. One of these specimens at a level just above the exit of the fifth nerve, which he has permitted us to examine, is quite demonstrative. There is a small area of degeneration just in front and a little to one side of the posterior longitudinal bundle. In a section at the level of the lower olive the area external and posterior to this body is less deeply stained. Bechterew⁴¹ is not able to give the superior termination of the central tegmental tract, but inferiorly he believes that it passes into the lower olive, and is in connection with the cerebellum by means of this body. Flechsig⁴² believes that the greater part of this tract is united with the lenticular nucleus by means of the ansa lenticularis, and the lower olive is in this way in direct connection with the lenticular nucleus. In congenital total defect of the cerebellum he has found the lower olives almost entirely absent; the central tegmental tracts completely absent, and the lenticular nuclei atrophic.

In a very recent article Bechterew⁴³ makes the statement that descending degeneration of the central tegmental tract has been repeatedly observed; he has observed it himself; and he believes that after section of its fibres the degeneration which occurs is downwards, and that the tract therefore represents a centrifugal system of fibres.

In our sections at the level of the entrance of the eighth nerves, there is a zone on either side less deeply stained, which we mention with some doubt as to its significance, between the superior olive and the fillet (see fig. 5). This corresponds exactly to the location of the central tegmental tract at this level, as described by Bechterew; but higher up we are not able to trace these atrophied fibres. This may be due to the fact that when nervous tissue has been destroyed early in life normal fibres have crowded into the region to replace those which have become degenerated; and this substitution is not always complete at all parts. The lenticular nucleus on each side is somewhat below the normal size, but the ansa lenticularis is not atrophied.

Deiters' cells are quite numerous. This need cause no surprise, for not only are they in connection with the

cerebellum by means of the direct sensory cerebellar tract of Edinger (Koelliker, *loc. cit.*, p. 344; Ferrier and Turner, *loc. cit.*, and others), but probably also with the vestibular nerves and the lateral columns of the cord (Monakow, Bechterew, *loc. cit.*, pp. 67 & 95).

The middle cerebellar peduncles degenerate after lesion of the cerebellum; they contain centrifugal fibres (Obersteiner, v. Bechterew, *loc. cit.*, 123). We can therefore readily understand the great diminution in number of these fibres which involves equally the stratum superficiale and the stratum profundum. The pyramidal tracts are not divided into the many separate bundles usually observed. Luciani (*loc. cit.*) has found that the middle peduncles arise chiefly in the middle lobe, but partly in the lateral. Koelliker says these fibres come almost exclusively from the cortex of the hemispheres (*loc. cit.*, p. 344). The nuclei pontis are almost entirely absent, and the cells of the nucleus reticularis tegmenti on each side are possibly not as numerous. The nuclei pontis are chiefly in connection with fibres from or to the opposite cerebellar hemisphere, although partly also with the fibres of the hemisphere of the same side; they are supposed to receive collaterals from the pyramidal fibres, and close union is thus made between the cerebrum and the contralateral side of the cerebellum (Obersteiner).⁴⁴ Cramer found the nucleus reticularis tegmenti on the side opposite the lesion partly atrophied, and he believes that his case proves the connection of this nucleus with the middle peduncle. Ferrier and Turner found atrophy of the fine fibres in this nucleus on the opposite side after extirpation of one cerebellar hemisphere, but no obvious affection of the cells; this is also confirmatory of Bechterew's view that some of the decussated fibres of the middle peduncle are in connection with cells in the tegmentum pontis.

The restiform bodies are small. The diminution is especially noticeable where these bodies are embraced by the two roots of the acoustic nerves. These bands contain chiefly fibres passing to the cerebellum. They were also atrophied in Cramer's case, as well as in others.

Atrophy of the posterior longitudinal bundles, of the direct cerebellar tracts, of the fillets, with the exception of the left lateral, of the periphery of the antero-lateral columns of the cord, and of the pyramidal tracts (Marchi)⁴⁵ is not to be observed in our case. Marchi's results were obtained on animals, and one must always be a little cautious in applying these directly to the human nervous system. But, on the other hand, our material did not permit the employment of his excellent method. Degeneration of the direct cerebellar tract downwards, even though it is a tract of ascending fibres, is not surprising when we remember that the optic tract, certainly sensory in function, contains fibres which terminate in the retina (Cajal), although some of these fibres are probably sympathetic (Mislowsky).⁴⁶

Moeli⁴⁷ found the lateral fillet diminished. Arndt was not able to observe in his case the degeneration of the posterior longitudinal bundles, of the fillets, of the periphery of the antero-lateral columns of the cord, and of the direct cerebellar tracts. Cramer noticed no change in the median fillet, but the lateral fillet of the atrophied side was not intact. The observations of Ferrier and Turner are entirely opposed to the statements of Marchi, viz., that removal of a lateral cerebellar lobe causes degeneration of the fillet and posterior longitudinal bundle. In none of their experiments were these structures altered, nor could they find descending degeneration in the antero-lateral columns or the anterior nerve-roots. Thomas,⁴⁸ on the other hand, has in large part confirmed the statements of Marchi, and Luciani has found the posterior longitudinal bundle degenerated after extirpation of one-half of the cerebellum.

Although the lower olives are much atrophied the median fillets appear normal, which is only confirmatory of the statements of Moeli and Marinesco,⁴⁹ viz., that the fillet has no connection with the lower olives.

In well-stained sections the pyramids are not degenerated. The corpus trapezoide is well defined and lies close to the periphery. The nuclei and fibres of the cranial nerves appear normal. In Hitzig's⁵⁰ case the trigeminus on the side corresponding to the normal cerebellar hemisphere, was

reduced one-half, and on the side corresponding to the atrophied hemisphere it was normal. The case which Redlich⁵¹ has recently reported is much in favour of the presence of fibres in the pyramidal tracts from the cerebellum. In many other respects his case is very similar to ours.

No marked changes are noted in the spinal cord, except in one portion of the middle to lower thoracic region, where the vessels in the grey matter at the base of one of the anterior horns are much distended; numerous hæmorrhages are seen, and the peri-vascular spaces are found infiltrated. Such changes are not to be noted elsewhere in the cord. The cells of Clarke's column are normal. The partial atrophy of this column and of the direct cerebellar tract, the diminution in the size of the anterior and posterior horns in the thoracic, and a part of the cervical region observed by Amaldi,⁵² in two cases of incomplete atrophy of the cerebellum—one from arrested development, the other from an early inflammatory process—are conditions which are not present in our case. Martinotti and Mercandino⁵³ examined five cases of cerebellar lesions in man, and obtained only negative results, as far as the cord was concerned. If there is any deficiency in any of the fibres of the cord we have no means of determining it, for when fibres degenerate early in infancy, or fail to develop, there is no formation of sclerotic tissue, and the cord at this part is merely a little smaller.

Examination of both the right and left frontal lobes has not disclosed anything remarkable; tangential fibres are present, perhaps less numerous, though this is always a difficult question to decide, and the cortex contains many cells. Cramer (*loc. cit.*) found in the frontal lobe of each side, after unilateral cerebellar atrophy, a slight decrease of fibres, especially of the tangential, and a slight increase of the neuroglia-cells. In the frontal lobe, opposite the atrophy, the ganglion-cells were a little smaller and the vessels were diseased.

A number of cases of cerebellar atrophy have been reported, in which the cerebrum was also diminished in size (Weber, Turner, Hitzig, Cramer, &c.). Turner was the

first, according to Luciani, who called attention to this, and he stated that the atrophied side of the cerebellum was opposite to the atrophied cerebral hemisphere. Turner believed that the atrophy of the cerebellum was secondary to the atrophy of the cerebrum, but cases of unilateral cerebral atrophy are known in which the cerebellum was not atrophied; this view must therefore be incorrect (Luciani).⁵⁴

On the other hand, the cerebellum has been found atrophied, and the cerebrum of normal size, as in Schultze's⁵⁵ case. There is probably a crossed connection between the cerebrum and cerebellum, by means of the anterior and middle cerebellar peduncles. Prof. and Madame Dejerine have shown that, though crossed hemiatrophy of the cerebellum from infantile cerebral hemiplegia is well known, the superior cerebellar peduncle in these cases is not degenerated, but merely atrophied, which means that it does not receive direct fibres from the cerebral cortex. They have shown the nature of the connection of the cerebral cortex with the superior peduncle. The destruction of the cerebellum in Luciani's experiments did not cause atrophy of the cerebrum. He believes the atrophy in the two parts is a simultaneous process due to a common cause. Arndt describes the connection of cerebrum with cerebellum as existing between the central gyri of one side, the internal capsule, the subthalamic region, the red nucleus, the opposite cerebellar peduncle, and the opposite cerebellar hemisphere. We have found the cerebrum considerably below the normal size, especially in the left hemisphere; but the two halves of the cerebellum were of equal size; the left thalamus was somewhat smaller than the right (see fig. 6). In Cramer's case the pulvinar opposite the cerebellar lesion was smaller. The lenticular nuclei are of about the same size.

This diminution in all parts of the central nervous system is in favour of the theory of hypoplasia. The small size of the posterior cranial fossæ is likewise confirmative. Brain and skull bear a close relationship to one another in their development, and, as Parker⁵⁷ says, "The type of fissuration

is due to the resultant forces produced by the interaction of the growth forces of the hemisphere combined with the pressure forces of the less rapidly expanding but symmetrically developing cavity of the skull. It would appear that during the earlier and even to the quite late stages of development, it is the brain which modifies the shape and structure of the skull." We have found the posterior fossæ but little larger than the cerebellum, and, as in Menzel's case, the cells of Purkinje, when present, are not shrivelled, which is a further proof of arrested development. Ziegler⁵⁸ considers a very small cerebellum as probably hypoplastic.

We have found the crura very small, and although at the level of the exit of the third nerves the internal band is well coloured, slightly higher up the fibres in this part are not numerous. The crura were also small in Menzel's case. In Arndt's case, there was a slight atrophy of the pyramidal fibres of the crura. The substantia nigra is normal. The ansa lenticularis on each side, as well as the anterior limb of the internal capsule, is well preserved.

Marie⁵⁹ gave the name of cerebellar hereditary ataxia in 1893 to a certain group of symptoms (*héréd-ataxie cérébelleuse*). He founded his description on the cases of Fraser,⁶⁰ Nonne,⁶¹ the remarkable series of Sanger Brown,⁶² and on those of Klippel and Durante,⁶³ and on the pathological findings in the first two of these reports. The important lesion is cerebellar atrophy, with simple diminution in the size of the spinal cord. Fraser considered his cases as arrested development. Nonne's case was due to the same condition, and the entire central nervous system was small. Friedreich's disease and cerebellar ataxia may be different forms of the same malady (Marie), and Menzel's case is much in favour of this view, as cord and cerebellum were both affected. It is, according to Menzel, the first case of Friedreich's disease reported in which the cerebellum was involved. Marie includes in the same group the family form of cerebral diplegia (Freud),⁶⁴ in which the spastic phenomena are more pronounced than in cerebellar hereditary ataxia, and in which slowness of speech, nystagmus, and

optic atrophy are found as in cerebellar hereditary ataxia; but it differs from the latter disease in greater spasticity, and in the absence of pseudo-tremor and incoördination.

Although lesions of the cerebellum may occur in Friedreich's disease, they are less prominent than in the cerebellar hereditary ataxia. Exaggeration of the knee-jerk is a sign of great value in distinguishing the latter from the malady of Friedreich (Marie, Brissaud, &c.). In Menzel's case the knee-jerk was preserved, and at first increased.

The chief characteristics of this disease, to which Marie gave a name, and which he fully described, are: heredity, age of development (between twenty and thirty, although occasionally seen in infancy), greater frequency in females and of transmission by them, slow development of the process, exaggeration of the patella-reflexes, gait like in Friedreich's disease, unstable erect station, no alteration of muscular force, absence of Romberg's sign, greater incoördination of movement in the lower extremities, fibrillary contractions, occasionally ankle-clonus, affections of the eyes (consisting of ptosis, nystagmus-like movements, frequent paralysis of the external recti, difficulty of convergence, imperfect reaction of the pupils, Argyll-Robertson's sign, rigidity of pupils, diminished visual acuity, amaurosis, changes in the papillæ), slow and explosive speech, absence of trophic disturbances (scoliosis, club-foot, &c.), death usually from an intercurrent long-affection (phthisis in one of Fraser's cases). Nonne considers this description given by Marie as very comprehensive. Let us now see how closely this last case of ours corresponds to this disease as far as the meagre clinical history permits us to judge. We have no history in favour of or against heredity, and there is no mention of trophic disturbance. We find development of the disease in early childhood (and in the boy of Fraser's two cases a slight reel or stagger was noticed about the age of two or three. The history of the beginning of the disease in the girl is said to be a repetition of that in the brother. In one of Nonne's three cases, incoördination began at the age of fourteen; in another it was noticed before the age of ten. This latter was the case in which an autopsy was



FIG. 5.

- a a* Areas faintly coloured and containing few nerve fibres.
- b* Superior olive.
- c* Centre of the facial nerve.
- d* Sclerotic area in left cerebellar hemisphere.

The section is cut obliquely. At a level a little higher than the one represented in the drawing the pyramids are enclosed by a few fibres of the middle cerebellar peduncles.

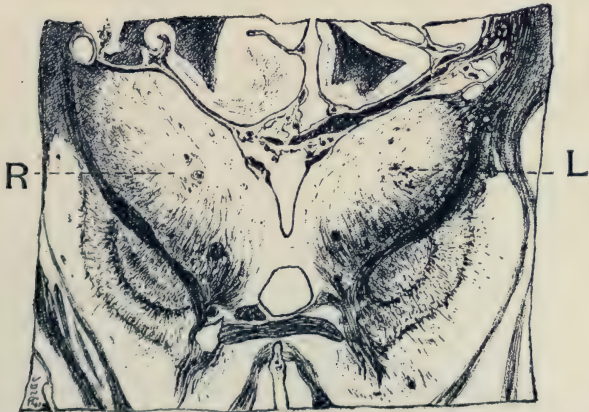


FIG. 6.

Horizontal section through the basal ganglia (*natural size*).

R Right.

L Left.

obtained, and the statement is made that as a child he was always weak). We find mention also of disturbance of gait (slight stagger) and of speech, defective eye-sight, paralysis of the left external rectus, dizziness, normal patellar reactions—though it is not at all impossible that these were somewhat exaggerated—mental disturbance, death from tuberculosis (which, though not of diagnostic value, is nevertheless interesting), and especially hypoplasia of high grade, with sclerosis of the cerebellum and simple diminution in the size of the spinal cord and brain. The case seems, therefore, to belong to the rare type described by Marie, although heredity cannot be established. Is every case in which many of the typical signs are present, and the required condition of the central nervous system is found after death, to be excluded from this classification when heredity cannot be demonstrated? As has been so often said, the disease must commence in some member of the family, and in our case we have no history of brothers and sisters. Higier,⁶⁵ in discussing the influence of heredity, does not think a characteristic symptom-complex should be excluded from a definite group of family diseases, merely because a similar condition cannot be shown in another member of the family. Our patient was able to preserve the erect position long enough to drill and steadily enough to have his photograph taken.

The family type of cerebellar disease is curiously seen in the report of Herringham and Andrewes.⁶⁶ Four kittens constituting one litter developed progressive weakness and tottering gait at different periods: two of these were examined and the cerebellum, especially the middle lobe, was found to be very small. In some of the cases of cerebellar sclerosis eye-symptoms were absent, and in the case of hereditary ataxia reported by Brissaud and Londe⁶⁷ scoliosis was present. One must be prepared to find transitional forms of cerebellar hereditary ataxia, and indeed Higier (*loc. cit.*) points out the close connection of this type with the cerebral diplegia. It seems to us perfectly proper to include the first case mentioned in this paper in this group.

Certain writers have emphasized the importance of

asymmetry in cerebellar lesions for the production of inco-ordinate movements (Vulpian, Schiff), while Nothnagel⁶⁸ regarded involvement of the vermis as necessary. Luciani,⁶⁹ judging from his experiments, believes the lateral lobes have as much importance functionally as the middle lobe, and although destruction of the lateral lobes may be latent longer than that of the vermis it is because a lesion of the same size in the latter location involves more peduncular fibres. When the symptoms were symmetrical on the two sides of the body in his experiments the destruction of the middle lobe was equal on the two sides. When the symptoms predominated on one side the lateral lobe of that side was particularly involved. Nothnagel's view, according to Luciani, cannot be correct, for he has found that the symptoms produced by destruction of the middle lobe may gradually disappear, which signifies that compensation is effected by other parts of the cerebellum. The theory that only asymmetrical lesions (Vulpian, Schiff) can cause motor disturbance, and that symmetrical lesions, even if extensive, may be latent, is also incorrect (Luciani). Symmetrical lesions cause symptoms of irritation and paralysis on both sides of the body, and the asymmetrical cause symptoms on both sides, but unequal in degree and extent (Luciani).

The cerebellum (Luciani) is functionally a homogenous organ, and the symptoms due to unilateral or bilateral, symmetrical or asymmetrical, limited or extensive lesions of the cerebellum are not different from one another in nature, but only in degree, extent and location. Latency is not dependent on location. In some cases it is due to slow development of the process and to compensation by other parts of the cerebellum, or even by parts outside of the cerebellum (Luciani). When the entire cerebellum has been affected careful examination has never failed to discover a greater or less degree of awkwardness of movement and instability of equilibrium (Ferrier, *loc. cit.*). Becker⁷⁰ mentions a number of cases of cerebellar diseases in which no symptoms had been present.

We have found the hypoplasia equal on the two sides of the cerebellum, and no statement has been made of pre-

dominance of symptoms on one side or the other. The vermis was sclerosed, and although both cerebellar hemispheres were hypoplastic, sclerosis was only found in a limited area in each hemisphere. It was a cerebellum in miniature, to use a borrowed expression.

One cannot read the literature on cerebellar disease without noticing the frequency of some infectious malady as the apparent cause of cerebellar atrophy or hypoplasia, or of Friedreich's disease. It is probable that such an infection acts as an "agent provocateur," as Hirschl⁷¹ suggests, on an already imperfectly developed nervous system, or increases morbid conditions already existing. Thus in Hirschl's case of Friedreich's disease the symptoms began after scarlet fever at the age of four, and were of slow progress until influenza attacked the patient fifteen years later. Hirschl reported, also, two cases of cerebellar hereditary ataxia in brothers, which developed after typhoid fever. Wagner⁷² also has reported two cases of Friedreich's disease in the same family; one developed after typhoid fever, the other after measles. Diphtheria and scarlet fever at the age of five caused the disease to be first noticed by the parents in the last case we have studied.

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A CASE WHICH AT THE AUTOPSY SHOWED EVIDENCE OF RECOVERY FROM A PRE- VIOUS ATTACK OF MENINGITIS.

BY W. HALE WHITE, M.D.

Physician to Guy's Hospital.

A MALE child, aged 13 months, was admitted into Guy's Hospital on January 2, 1896. Clinical Clerk, Mr. C. J. Harnett, M.B. There is no history of tubercle in the family, nor of ear trouble or syphilis in the child. The parents state that he was quite well till three weeks ago, when he began to be sick after food, and was fretful. A week later he had a fit, which the mother describes as follows:—The arms and legs became suddenly flexed and fixed. There was violent twitching of the right side of the face. This fit lasted about twelve minutes, and after it the child became very drowsy. Since this he has had many fits, on some days there have been twelve in twenty-four hours. Each fit consists of convulsions of all four extremities, and lasts about ten minutes. After it the child is again drowsy. He has not been sick.

On Admission.—The head is large and the fontanelles are rather widely open and bulging. The temperature is 100·2° F. and the pulse 140. The child seems ill and takes no notice of anything. The optic discs are healthy and no paralysis is noticeable. The thoracic and abdominal organs appear healthy.

January 3.—To-day the head is markedly retracted and the child has been observed several times to rigidly extend its limbs. During these fits the hands are clenched tightly, and the patient perspires profusely on the face, but this profuse perspiration is invariably strictly confined to the left side of the face, the line of

demarcation between the dry and the moist sides extending straight down the middle of the face, so that one side of the nose will be covered with beads of perspiration when the other is perfectly dry. The left arm was always moister than the right. The temperature in the left axilla was 100.2° , when that in the right was 100° . In many of the above described fits there were convulsive movements confined to the left arm and leg. The breathing was often distinctly of the Cheyne-Stokes type.

January 4.—The patient lies in the same drowsy condition. The pulse is slow, the breathing irregular. The right side of the face does not move quite so well as the left. The head is retracted. There are frequent irregular movements of the right arm. The pupils are widely dilated, and there is definite commencing optic neuritis. The unilateral sweating is still observable.

January 5.—The patient is in the same condition. The optic neuritis is more marked.

January 6.—The child still has well-marked Cheyne-Stokes breathing. At the commencement of each phase in which the breathing increases in rapidity the child utters a cry, the left lower eyelid twitches, and the mouth is drawn over to the left. The right arm becomes rigid in a position of extension, and then is alternately flexed and extended, the fingers being clenched into the palm of the hand. The head is still more retracted, and the face is turned to the right. The spinal muscles become rigid, especially on the right, so that the back is arched, and the body is bent to the right. The muscles of the legs, too, are firmly contracted, so that the body rests on the head and the heels. The sweating is still unilateral. The pulse has become 168, the temperature 104.4° . The optic neuritis is very well marked. The child died at 5.30 p.m. At the autopsy there was much recent basal meningitis. In each fissure of Sylvius about half an inch from its commencement was a caseous tubercular nodule, the size of a very large pea. Both these nodules were in the meninges, and could be easily freed from the brain. No other tuberculous deposit was found anywhere. The rest of the body was healthy.

Although most physicians would, I think, be prepared to admit that meningitis is, in certain rare cases, capable of recovery yet when the patient gets well there is always a certain element of doubt in the case. Therefore, it seemed to me that this case was worthy of record, for the presence of two old caseous tubercular nodules in the meninges as well as recent acute inflamma-

tion of them shows that there must have been some meningitis previous to that which killed the child, unless we believe that they represented an earlier stage of the same attack as that which was fatal, but the history seems too short for this explanation, for the caseous nodules looked as though they must be more than a month old.

Rules

AND

LIST OF OFFICE BEARERS AND MEMBERS

OF THE

Neurological Society of London, 1896,

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1895.

LONDON

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1896

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Neurological Society of London.

REPORT OF COUNCIL.

JANUARY 16, 1896.

The Council has the pleasure to report the continued success and prosperity of the Society. The members now number 166.

The Inaugural Address was delivered at the Annual General Meeting on January, 7th, by the President, Sir WILLIAM BROADBENT, Bart., on "Some Speculations on the Constitution and Action of the Nervous System."

The proceedings appended hereto, show the character and value of the work of the Society during the past year.

The Council deeply regret the loss sustained by the Society by the deaths of Dr. BRISTOWE, F.R.S., Past President, and of Dr. HACK TUKE, Vice-President, both of whom were also Past Treasurers.

Proceedings of the Society during the year 1895.

(A complete list of the proceedings from the foundation of the Society to the year 1893 inclusive, will be found in *Brain*, vol. xvii., p. 19 of the Report.)

January 17th, Sir WILLIAM H. BROADBENT, Bart., Inaugural address, "Some Speculations on the Constitution and Action of the Nervous System," *Brain*, vol. xviii., p. 186.

March 7th, Dr. WALLER, F.R.S., "The Weber-Fechner Law as Illustrated by Muscle, Nerve and Retina," *Brain*, vol. xviii., p. 200. Drs. SHERRINGTON, F.R.S., and MOTT, "The Influence of Sensation upon Movement," *Proc. Roy. Soc.*, 1895.

April 25th, Dr. ANDRIEZEN, "The Pathology of Alcoholic Insanity." Dr. TOOTH, "A Shrunk Brain from a Case of Chronic Alcoholism."

June 6th, Dr. DONKIN, "Two Cases of Friedreich's Disease and a Case of Chorea with Anæsthesia." Mr. LUNN, Dr. BEEVOR, and Mr. BALLANCE, "A Case of Successful Removal of Tumour of the Cerebellum." Drs. PENROSE and BEEVOR, "A Case of Sclerodermia." Dr. BEEVOR for Dr. TAYLOR, "A Case of Sclerodermia." Dr. HALE WHITE (1) "A Case of Respiration by the Diaphragm alone;" (2) "A Case of Trephining four times for Cerebral Tumour, each time with Relief of Symptoms;" (3) "A Case of Probable Osteitis Deformans." Mr. HARRIS, for the PRESIDENT, "A Case of Primary Progressive Myopathy, in which Pseudo-Hypertrophy and Atrophy Co-existed."

July 11th, Dr. COLMAN, "Cerebellar Symptoms in a Cat, Atrophy of Cerebellum demonstrated *post-mortem*." Dr. RUSSELL, "Congenital Defect of Cerebellum in Dog" (lantern demonstration). Dr. TURNER, (1) "Tumour of One Lobe of Cerebellum;" (2) "Syringomyelia and Glioma" (projection microscope). Dr. HUBERT BRISTOWE, "A Case of so-called Heterotopia of the Spinal Cord." Dr. PITT, "Degeneration of Posterior Columns in Pernicious Anæmia, two Cases" (projection microscope). Dr. PANDI (of Buda-Pesth), "Spinal Cord in Pseudo-tabes, Arteriosclerosis, and Diabetes." Dr. MOTT, "Preliminary Communication upon the Chemistry of Degeneration" (lantern demonstration).

Nov. 7th, Mr. JONATHAN HUTCHINSON, F.R.S., "Two Cases of almost Universal Sclerodermia." Dr. CROCKER (introduced by the PRESIDENT), (1) "Two Cases of Supra-orbital Morphœa;" (2) "A Case of Whitlow of thirty-five Years' standing;" (3) "A Case of Infantile Paralysis with inflammatory Lesion of the Skin." Dr. BEEVOR, "A Case of Facial Hemiatrophy." Dr. GALLOWAY (introduced by the PRESIDENT), (1) "A Case of Supra-orbital Morphœa;" (2) "A Case of Syringomyelia with appearances of Acromegaly." Dr. MENZIES, "A Case of Herpes Labialis, followed by Paralysis of flexor muscles of forearm and hand and Œdema." Dr. CONNOR (by Mr. Hutchinson), "A Case of Hygrostomia in an Edentulous Woman." Dr. SHUTTLEWORTH, "Adenoma Sebaceum in Imbeciles" (photographs).

BALANCE SHEET, 1895.

NEUROLOGICAL SOCIETY.

RECEIPTS.				EXPENDITURE.	
		£	s. d.		£ s. d.
To Balance in hand	141	8 8	By Stationery—Pulman & Sons	15 9 11
Subscriptions for current year	153	1 0	Printing—Bale & Sons ...	3 9 1
Do. in advance...	...	2	0 0	Illustrations—Danielsson	53 17 0
Do. in arrears	10	0 0	Do. Bale & Sons	1 15 6
Share of profit on "Brain"	9	15 3	Do. Thévoz & Co.	31 0 0
Donation from Sir W. Broadbent for indexing "Brain"	37	16 0	Do. Briginshaw	10 10 0
				Microscopes—R. and J. Beck	4 12 0
				Hire of Rooms and Refreshments—	
				Royal Medico-Chirurgical Society	15 8 0
				Messrs. Macmillan	93 7 3
				Lantern Expenses	1 0 0
				Petty Cash	1 14 6
				Excess Subscription	1 0 0
				Bank Charges	0 2 6
					£ s. d.
				Balance ...	82 19 2
				Donation ...	37 16 0
				(From Sir William Broadbent)	
				Total Balance ...	120 15 2
					£354 0 11

*Audited and found correct, (JAMES TAYLOR,
December 13th, 1895.) J. S. RISIEN RUSSELL.*

CHARLES E. BEEVOR, *Hon. Treasurer.*

RULES.

- 1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.
- 2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.
- 3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.
- 4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.
- 5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.
- 6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.
- 7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.
- 8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.
- 9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.
- 10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of a meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a Special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watterville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. HERBERT SPENCER, London.
 Professor WUNDT, Leipzig.
 Professor DU BOIS REYMOND, Berlin.

CORRESPONDING MEMBERS.

Professor ERB, Leipzig.
 „ GOLTZ, Strasburg.
 „ GRASSET, Montpellier.
 „ HITZIG, Halle.
 „ JOLLY, Berlin.
 „ LUCIANI, Rome.
 „ MUNK, Berlin.
 „ OBERSTEINER, Vienna.
 „ RIBOT, Paris.
 „ SCHIFF, Geneva.
 Dr. WEIR MITCHELL, Philadelphia.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. HUGHLINGS JACKSON, M.D., F.R.S.
 1887 SAMUEL WILKS, M.D., F.R.S.
 1888 Sir JAMES CRICHTON-BROWNE, M.D., F.R.S.
 1889 JONATHAN HUTCHINSON, F.R.S.
 1890 THOMAS BUZZARD, M.D.
 1891 JOHN S. BRISTOWE, M.D., F.R.S.
 1892 HENRY C. BASTIAN, M.D., F.R.S.
 1893 EDWARD A. SCHÄFER, F.R.S.
 1894 DAVID FERRIER, M.D., F.R.S.
 1895 Sir WILLIAM H. BROADBENT, Bart., M.D.
 1896 ALEXANDER HILL, M.A., M.D.

* * Members are requested to communicate with the Secretaries when corrections are necessary.

—:O:—

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.	Tr., Treasurer.
Pres., President.	Sec., Secretary.
V.P., Vice-President.	C., Member of Council.

ORDINARY MEMBERS.

Elected.

- 1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.
 1894 ANDRIEZEN, W. LLOYD, M.D., West Riding Asylum, Wakefield.
 1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W.
 O.M. BARLOW, THOMAS, M.D., F.R.C.P., 10, Wimpole Street, W.
 „ BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5.)
 „ BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.
 „ BEEVOR, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)
 „ BENNETT, A. HUGHES, M.D., F.R.C.P., 76, Wimpole Street, W. (Sec., 1886-7; C., 1888-91.)
 1888 BIRT, ERNEST, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.
 1892 BOWLBY, ANTHONY A., F.R.C.S., 24, Manchester Square, W.
 1893 BOWMAN, HENRY MOORE, M.D., M.R.C.P., 21, Welbeck Street, W.
 1889 BRADFORD, J. R., M.D., D.Sc., M.R.C.P., F.R.S., 52, Upper Berkeley Street, W.
 O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)
 1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.
 1892 BRISTOWE, HUBERT CARPENTER, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.
 O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1895.)
 1894 BRODIE, T. GREGOR, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.
 1888 BROWN, SANGER, M.D., 34, Washington Street, Chicago.
 O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C. 1895.)
 O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.
 „ BRUNTON, T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896.)

- 1890 BURY, JUDSON SYKES, M.D., M.R.C.P., 10, St. John Street, Manchester.
- O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-5.)
- 1891 CAGNEY, JAMES, M.D., M.Ch., M.R.C.P., 93, Wimpole Street, W.
- 1889 CARTER, ROBERT BRUDENELL, F.R.C.S., 31, Harley Street, W.
- 1887 CATTELL, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.
- 1891 CLARKE, J. MITCHELL, M.D., M.R.C.P., 28, Pembroke Road, Clifton, Bristol.
- O.M. COBBOLD, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.
- 1892 COLE, ROBERT HENRY, M.B., L.R.C.P., M.R.C.S., Moorcroft, Hillingdon, Uxbridge.
- 1890 COLMAN, W. S., M.D., M.R.C.P., 22, Wimpole Street, W.
- „ CORNER, HARRY, M.D., L.R.C.P., Earlswood Asylum, Redhill, Surrey.
- 1894 CUMING, JAMES, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.
- 1895 DAVIDSON, ANDREW, M.B., C.M., Winson Green Asylum, Birmingham.
- 1894 DEAN, H. PERCY, M.D., F.R.C.S., 84, Wimpole Street, W.
- O.M. DONKIN, H. B., M.B., F.R.C.P., 108, Harley Street, W.
- „ DOWN, J. LANGDON, M.D., F.R.C.P., 81, Harley Street, W.
- „ DRESCHFELD, JULIUS, M.D., F.R.C.P., 325, Oxford Road, Manchester.
- „ DRUMMOND, DAVID, M.D., 6, Saville Place, Newcastle-on-Tyne.
- 1892 DUPUY, EUGENE, M.D., 53, Avenue Montaigne, Paris.
- 1889 ECCLES, A. SIMONS, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.
- O.M. EDMUNDS, WALTER, M.B., F.R.C.S., 75, Lambeth Palace Road, S.E.
- „ FERRIER, DAVID, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)
- „ FOSTER, MICHAEL, M.D., F.R.S., Shelford, Cambridge.
- 1888 FOX, ARTHUR, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.
- O.M. FOX, E. LONG, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.
- „ GASKELL, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (C., 1896.)
- „ GEE, SAMUEL J., M.D., F.R.C.P., 31, Upper Brook Street, W.
- „ GODLEE, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.
- 1890 GOODALL, E. W., M.D., Eastern Hospitals, Homerton, E.
- 1892 GOTCH, FRANCIS, M.A., F.R.S., 11, Prince's Park, Liverpool.
- 1893 GRANT, RUNDAS, M.D., F.R.C.S., 8, Upper Wimpole Street, W.
- O.M. GUNN, R. MARCUS, M.B., F.R.C.S., 54, Queen Anne Street, W.
- 1895 GUTHRIE, LEONARD, M.D., M.R.C.P., 15, Upper Berkeley Street, W.
- 1893 HALLIBURTON, WILLIAM DOBINSON, M.D., F.R.C.P., F.R.S., 9, Ridgmount Gardens, W.C.
- O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.
- 1889 HANDFORD, HENRY, M.D., M.R.C.P., 14, Regent Street, Nottingham.
- 1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 109, Harley Street, W.
- O.M. HAYCRAFT, J. B., M.B., F.R.S.E., The University, Cardiff.

- 1891 HEAD, HENRY, M.D., C.M., L.R.C.P., 6, Clarence Terrace, Regent's Park, N.W.
- O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres. 1896 ; V.P., 1895 ; C., 1892-94.)
- „ HOPKINS, JOHN, F.R.C.S., London Sick Asylum, Cleveland Street, W.
- „ HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (V.P., 1896 ; C., 1892-3.)
- 1889 HUGGARD, WILLIAM R., M.D., M.R.C.P., Davos Platz, Switzerland.
- 1896 HULME, GEORGE FREDERICK, M.B., M.S.Ed., Montague Road, Felixstowe.
- 1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., 3, Trinity Street, Cambridge.
- O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889 ; V.P., 1887-88 ; C., 1886, 1890.)
- 1889 HYSLOP, T. B., M.D., C.M., Bethlem Royal Hospital, Lambeth, S.E.
- O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886 ; C., 1887-92.)
- „ JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.
- 1895 JOHNSTON, GEORGE, M.B., M.R.C.P., 2, Brunswick Place, Brighton.
- 1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 9, Upper Wimpole Street, W.
- 1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.
- „ KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.
- 1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.
- O.M. LANGLEY, J. N., M.A., F.R.S., Trinity College, Cambridge.
- 1888 LAWFORD, J. B., M.D., F.R.C.S., 55, Queen Anne Street, W.
- O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.
- 1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.
- „ MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 59, Welbeck Street, W.
- O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896.)
- 1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.
- O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93 ; C., 1893-96.)
- „ MANN, J. DIXON, M.D., F.R.C.P., 16, St. John Street, Manchester.
- 1894 MARTIN, SIDNEY, M.D., F.R.C.P., 10, Mansfield Street, W.
- 1893 MAUDE, ARTHUR, L.R.C.P., M.R.C.S., Westerham, Kent.
- 1891 MAY, W. PAGE, M.D., M.R.C.S., Goldburn, Eltham Road, Blackheath.
- 1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.
- O.M. MERCIER, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.
- „ MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895.)
- 1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.
- O.M. MORRIS, MALCOLM, F.R.C.S.Ed, 8, Harley Street, W.

- 1888 MOTT, F. W., M.D., F.R.C.P., 84, Wimpole Street, Cavendish Square, W. (Sec., 1894.)
- 1894 MURRAY, GEORGE, M.B., M.R.C.P., 2 Saville Place, Newcastle-on-Tyne.
- 1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 27, Savile Row, W.
- O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.
- „ NICOLSON, DAVID, M.D., M.R.C.P.Ed., Broadmoor, Wokingham, Berks.
- 1888 NIERMEYER, J. H. H., M.D., Amsterdam.
- „ NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.
- 1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.
- 1887 OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.
- „ OLIVER, THOMAS, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.
- O.M. ORANGE, WM., C.B., M.D., F.R.C.P., 12, Lexham Gardens, Kensington, W.
- „ ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)
- „ ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895.)
- „ PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)
- 1895 PINDER, GEORGE, M.B., B.C., Seafield House, Ramsey, Isle of Man.
- 1888 PITT, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.
- O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 30, Wimpole Street, W.
- 1887 PRINGLE, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.
- O.M. PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.
- 1894 RANSOM, WILLIAM B., M.D., M.R.C.P., The Pavement, Nottingham.
- 1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 2, Harley Street, W.
- O.M. REID, E. W., M.B., University College, Dundee.
- 1893 RENNIE, GEORGE EL., M.D., 16, College Street, Sydney, N. S. Wales.
- 1895 REYNOLDS, ERNEST S., M.D., M.R.C.P., 23, St. John Street, Manchester.
- 1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.
- 1891 RIVERS, W. H. R., M.D., M.R.C.S., St. John's College, Cambridge.
- 1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.
- „ RUFFER, MARC ARMAND, M.D., 5, York Terrace, Regent's Park, N.W.
- 1889 RUSSELL, J. S. R., M.D., C.M., M.R.C.P., 4, Queen Anne Street, W. (Sec., 1896.)
- O.M. SANDERSON, J. BURDON, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.
- „ SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (V.P., 1895; C., 1886-90.)
- 1888 SAVILL, T. D., M.D., M.R.C.P., 12, Upper Berkeley Street, W.
- O.M. SCHÄFER, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)
- „ SEMON, FELIX, M.D., F.R.C.P., 39, Wimpole Street, W.
- „ SHARKEY, S. J., M.D., F.R.C.P., 2, Portland Place, W. (C., 1889-92.)
- 1892 SHAW, JAMES, M.D., M.Ch., Donard House, Kensington, Liverpool.

- O.M. SHERRINGTON, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)
- 1894 SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond.
- 1895 SMITH, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.
- 1887 SMITH, R. PERCY, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.
- 1895 SMITH, TELFORD, M.D., B.S., Royal Albert Asylum, Lancaster.
- 1895 STANSFIELD, THOS. E. K., M.B., 7, Claybury Asylum, Woodford, Essex.
- 1892 STARLING, ERNEST HENRY, M.D., M.R.C.P., B.S., 107, Clifton Hill, N.W.
- O.M. STEWART, Sir T. GRAINGER, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.
- „ STIRLING, WM., M.D., D.Sc., Owens College, Manchester.
- 1895 STOUT, G. F., M.A., St. John's College, Cambridge.
- 1887 SUCKLING, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.
- O.M. SULLY, JAMES, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)
- 1889 SUTHERLAND, HENRY, M.D., M.R.C.P., 6, Richmond Terrace Whitehall.
- 1888 SYERS, HENRY W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.
- 1891 TAYLOR, JAMES, M.D., M.R.C.P., 49, Welbeck Street, W.
- 1889 THORBURN, WM., F.R.C.S., Rusholme, Manchester.
- 1892 TITCHENER, E.B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.
- O.M. TOOTH, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95 C., 1896.)
- 1892 TREVELYAN, E. F., M.D., BSc., 40, Park Square, Leeds.
- „ TUCKEY, CHARLES LLOYD, M.D., C.M., 14, Green Street, Grosvenor Square, W.
- 1892 TUKE, J. BATTY, M.D., F.R.C.P.Ed., 20, Charlotte Square, Edinburgh.
- 1891 TUKE, THOMAS SEYMOUR, M.B., M.R.C.S., Chiswick House, Chiswick.
- 1891 TURNER, WILLIAM ALDREN, M.D., M.R.C.P., 13, Queen Anne Street, W.
- O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.
- 1888 VOORTHUIS, J. A., M.D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)
- 1895 WADE, ARTHUR L., M.D., Somerset and Bath Asylum, Wells.
- O.M. WALLER, AUGUSTUS M.D., F.R.S., 16, Grove End Road, N.W. (C., 1894.)
- 1894 WALKER, A. STODART, M.B., 30, Walker Street, Edinburgh.
- O.M. WARD, J., D.Sc., Trinity Collage, Cambridge.
- „ WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.
- 1892 WASHBOURN, J. W., M.D., F.R.C.P., Guy's Hospital, S.E.
- O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95 ; Sec. 1886-9 ; Editor of *Brain*.)

- O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley Street, W.
- 1894 WHITING, ARTHUR J., M.D., National Hospital, Queen Square, W.C.
- 1889 WIGGLESWORTH, JOSEPH, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.
- 1894 WILLIAMSON, RICHARD T., M.D., M.R.C.P., 294, Oxford Road, Manchester.
- O.M. WILKS, SAMUEL, M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)
- 1893 WILLS, ERNEST, M.D., M.R.C.P., Claybury Asylum, Woodford Bridge, Essex.
- „ WOOD, GUY M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.
- 1889 WOOD, T. OUTTERSON, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.
- 1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.



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